passmedicine PART 2

THERAPUTICS

2024

A peri-arrest call is put out for a 71-year-old woman on the high dependency unit. An arterial line was being inserted and upon administration of some local anaesthetic, a sudden reduction in consciousness was noted. The patient has now started to have a seizure.

Her vital signs are 91% on 15 litres of oxygen, respiratory rate of 7/min, heart rate of 54/min, blood pressure of 72/54mmHg, the patient is unresponsive and has a temperature of 35.9ºC.

As well as emergency management, what would be the most effective treatment to give in this instance?

- Atropine
- Flumazenil
- Fomepizole
- Lipid emulsion
- Sugammadex

Submit answer

Reference ranges

Score: **0%**

1 -

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Her vital signs are 91% on 15 litres of oxygen, respiratory rate of 7/min, heart rate of 54/min, blood pressure of 72/54mmHg, the patient is unresponsive and has a temperature of 35.9ºC.

As well as emergency management, what would be the most effective treatment to give in this instance?

Atropine	6%
Flumazenil	7%
Fomepizole	2%
Lipid emulsion	81%
Sugammadex	4%

Local anesthetic toxicity can be treated with IV 20% lipid emulsion

Important for me Less important

This woman is suffering from local anaesthetic toxicity. The most common symptoms initially are confusion, drowsiness, and dizziness, however, this can progress to seizures, respiratory arrest, and coma. Treatment includes life support measures and benzodiazepines to control seizures. The most appropriate reversal agent is intravenous lipid emulsion which is, therefore, the correct answer.

Atropine is effective at treating bradycardia however is not the reversal agent for local anaesthetic toxicity. Whilst this woman is bradycardic, this is very likely to be refractory to treatment solely with lipid emulsion without co-administration of atropine.

Flumazenil is the reversal agent for benzodiazepine overdoses however has no role in local anaesthetic toxicity. In this instance, benzodiazepines are indicated to control the seizures and flumazenil would only act to further lower the seizure threshold.

Fomepizole is the reversal agent for ethylene glycol or methanol which can cause reduced consciousness but has no role in local anaesthetic toxicity which is the diagnosis here.

Sugammadex is the reversal agent for muscle relaxants such as rocuronium. Muscle relaxants are not administered during the insertion of an arterial line and therefore sugammadex is not indicated here.

Discuss (2) Improve

Next question

Local anaesthetic agents

Lidocaine

- An amide
- Local anaesthetic and a less commonly used antiarrhythmic (affects Na channels in the axon)
- Hepatic metabolism, protein bound, renally excreted
- Toxicity: due to IV or excess administration. Increased risk if liver dysfunction or low protein states. Note acidosis causes lidocaine to detach from protein binding. Local anesthetic toxicity can be treated with IV 20% lipid emulsion
- Drug interactions: Beta blockers, ciprofloxacin, phenytoin
- Features of toxicity: Initial CNS over activity then depression as lidocaine initially blocks inhibitory pathways then blocks both inhibitory and activating pathways. Cardiac arrhythmias.
- Increased doses may be used when combined with adrenaline to limit systemic absorption.

Cocaine

- Pure cocaine is a salt, usually cocaine hydrochloride. It is supplied for local anaesthetic purposes as a paste.
- It is supplied for clinical use in concentrations of 4 and 10%. It may be applied topically to the nasal mucosa. It has a rapid onset of action and has the additional advantage of causing marked vasoconstriction.
- It is lipophillic and will readily cross the blood brain barrier. Its systemic effects also include cardiac arrhythmias and tachycardia.
- Apart from its limited use in ENT surgery it is otherwise used rarely in mainstream surgical practice.

Bupivacaine

- Bupivacaine binds to the intracellular portion of sodium channels and blocks sodium influx into nerve cells, which prevents depolarization.

- It has a much longer duration of action than lignocaine and this is of use in that it may be used for topical wound infiltration at the conclusion of surgical procedures with long duration analgesic effect.
- It is cardiotoxic and is therefore contra indicated in regional blockage in case the tourniquet fails.
- Levobupivicaine (Chirocaine) is less cardiotoxic and causes less vasodilation.

Prilocaine

- Similar mechanism of action to other local anaesthetic agents. However, it is far less cardiotoxic and is therefore the agent of choice for intravenous regional anaesthesia e.g. Biers Block.

All local anaesthetic agents dissociate in tissues and this contributes to their therapeutic effect. The dissociation constant shifts in tissues that are acidic e.g. where an abscess is present, and this reduces the efficacy.

Doses of local anaesthetics

Agent	Dose plain	Dose with adrenaline
Lignocaine	3mg/Kg	7mg/Kg
Bupivacaine	2mg/Kg	2mg/Kg
Prilocaine	6mg/Kg	9mg/Kg

These are a guide only as actual doses depend on site of administration, tissue vascularity and co-morbidities.

Maximum total local anaesthetic doses

- Lignocaine 1% plain - 3mg/ Kg - 200mg (20ml)
- Lignocaine 1% with 1 in 200,000 adrenaline - 7mg/Kg - 500mg (50ml)
- Bupivicaine 0.5% - 2mg/kg- 150mg (30ml)

Maximum doses are based on ideal body weight

Effects of adrenaline

Adrenaline may be added to local anaesthetic drugs. It prolongs the duration of action at the site of injection and permits usage of higher doses (see above). It is contra indicated in patients taking MAOI's or tricyclic antidepressants. The toxicity of bupivacaine is related to protein binding and addition of adrenaline to this drug does not permit increases in the total dose of bupivacaine, in contrast to the situation with lignocaine.

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Local anesthetics

Osmosis - YouTube 6 0

General & local anaesthetics

Speed Pharmacology - YouTube 2 0

Local anaesthetic toxicity

Kloss and Bruce - YouTube 4 1

Report broken media

Score: **15.1%**

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A 24-year-old man with a history of depression and self-harm is brought to the emergency department by a friend 2 hours after an impulsive overdose of ferrous sulphate. He weighs 65kg and has ingested 65 x 200mg ferrous sulphate tablets. This is equivalent to 4225mg of elemental iron (65mg/kg).

On assessment, he denies any abdominal pain, diarrhoea or vomiting.

Observations are as follows:

- Temperature 36.5
- Respiratory rate 18/min
- Saturations 98% on air
- Heart rate 82bpm
- Blood pressure 136/74 mmHg

Venous blood results show:

Hb	133 g/l	Na^+	139 mmol/l	Bilirubin	8 µmol/l
Platelets	287 * 10^9/l	K^+	4.1 mmol/l	ALP	89 u/l
WBC	8.3 * 10^9/l	Urea	3.4 mmol/l	ALT	34 u/l
Neuts	6.2 * 10^9/l	Creatinine	72 µmol/l	Albumin	40 g/l
Lymphs	1.9 * 10^9/l			Serum iron	70 umol/l
Eosin	0.2 * 10^9/l			pH	7.40

Abdominal x-ray demonstrates the presence of iron in the stomach.

What is the appropriate initial medical management

- Whole bowel irrigation
- Activated charcoal
- Emergency endoscopy for removal of iron from the stomach
- Observe and repeat iron levels 4-6 hours after ingestion

Administer desferrioxamine

Submit answer

Reference ranges

Score: **0%**

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2 -

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Lymphs	1.9 * 10^9/l			Serum iron	70 umol/l
Eosin	0.2 * 10^9/l			pH	7.40

Abdominal x-ray demonstrates the presence of iron in the stomach.

What is the appropriate initial medical management

Whole bowel irrigation	28%
Activated charcoal	24%
Emergency endoscopy for removal of iron from the stomach	9%
Observe and repeat iron levels 4-6 hours after ingestion	7%

Administer desferrioxamine 33%

Discuss (8) Improve

Next question

Iron overdose

Iron overdose is potentially very serious. Major complications that can occur include:

- Metabolic acidosis
- Erosion of gastric mucosa → GI bleeding
- Shock
- Hepatotoxicity and coagulopathy

Management is guided by the total amount of iron ingested (elemental iron/kg) and the presence/absence of symptoms (abdominal pain, diarrhoea, vomiting, lethargy).

Patients who have ingested less than 40mg/kg elemental iron and are asymptomatic can be observed at home.

Patients who have ingested > 40mg/kg elemental iron or who are symptomatic need medical assessment with serum iron levels measured 2-4 hours post-ingestion and abdominal x-ray.

Whole bowel irrigation is the decontamination procedure of choice and is performed on all patients presenting within 4 hours who have ingested > 60mg/kg elemental iron or have undissolved tablets on abdominal x-ray.

Activated charcoal is ineffective in iron poisoning.

Desferrioxamine is indicated in:

- Patients with serum iron level > 90umol/l,
- Patients with serum iron level 60-90umol/l, who are symptomatic or have persistent iron on abdominal x-ray despite whole bowel irrigation
- Any patient with shock, coma or metabolic acidosis

Endoscopy or surgery may be required if whole bowel irrigation is not effective or iron is adhered to the gastric wall.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 31-year-old gentleman, with known ulcerative colitis, presents with a three-day history of profuse bloody diarrhoea. He reports opening his bowels up to 12 times each day and describes passing both bloody stool and frank blood.

On examination he is pale. His heart rate is 95 beats per minute, respiratory rate 16 breaths per minute, blood pressure 128/78 mmHg. His chest is clear to auscultation and his abdomen is soft but diffusely tender. The patient is ambulant and despite his diarrhoea appears relatively well.

The patient is diagnosed with a flare of his ulcerative colitis. Intravenous access is obtained and fluids and intravenous steroids given.

Admission bloods show:

Hb	102 g/l
Platelets	556 * 10^9/l
WBC	17.5 * 10^9/l
ESR	78 mm/h

Regarding his venous thromboembolism prophylaxis, what should happen during this admission?

- Compression stockings only
- No prophylaxis due to active bleeding
- Intermittent pneumatic compression devices
- Prophylactic dose low molecular weight heparin and compression stockings
- Treatment dose low molecular weight heparin and compression stockings

Submit answer

Reference ranges

A 31-year-old gentleman, with known ulcerative colitis, presents with a three-day history of profuse bloody diarrhoea. He reports opening his bowels up to 12 times each day and describes passing both bloody stool and frank blood.

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Regarding his venous thromboembolism prophylaxis, what should happen during this admission?

Compression stockings only	22%
No prophylaxis due to active bleeding	7%
Intermittent pneumatic compression devices	20%
Prophylactic dose low molecular weight heparin and compression stockings	49%
Treatment dose low molecular weight heparin and compression stockings	2%

The severity of a flare of ulcerative colitis can be categorised used the Truelove and Witt criteria:

SEVERITY	MILD	MODERATE	SEVERE
Bowel movements per day	fewer than 4	4-6	>6 and features of systemic upset
Blood in stools	Small amounts	Between mild and severe	Visible blood

Pyrexia (temp >37.8ºC)	No	No	Yes
Pulse >90 bpm	No	No	Yes
Anaemia	No	No	Yes
ESR (mm/hr)	30 or below	30 or below	Above 30

Based on these criteria, this patient can be defined as suffering an acute severe flare of ulcerative colitis which is a medical emergency. Intensive treatment with intravenous steroids and early surgical intervention has reduced the mortality from acute severe ulcerative colitis to 2.9% however the colectomy rate of 30% in acute flares has not changed for 40 years.

Whilst this patient is complaining of passing blood per rectum an acute flare of ulcerative colitis is an extremely prothrombotic state and as a result, the British Society of Gastroenterology recommends that all patients should receive subcutaneous heparin to reduce the risk of thromboembolism. Intermittent pneumatic compression devices are a form of venous thromboembolism prevention that has evidence in acute stroke so is not the correct answer here. Whilst the patient is in a prothrombotic state, there is no indication for treatment dose low molecular weight heparin and this would increase the patient's risk of excessive blood loss.

The full guideline from the British Society of Gastroenterology can be found here: http://www.bsg.org.uk/images/stories/docs/clinical/guidelines/ibd/ibd2011.pdf

Discuss (6) Improve

Next question >

Venous thromboembolism: prophylaxis in patients admitted to hospital ★

VTEs can cause severe morbidity and mortality, but they are preventable. Current NICE guidelines (updated for 2018) outline recommendations for assessment and management of patients at risk of VTE in hospital.

Risk factors

All patients admitted to hospital should be individually assessed to identify risk factors for VTE development and bleeding risk. For medical and surgical patients the recommended risk proforma is the department of healths VTE risk assessment tool.

The following inpatients would be deemed at increased risk of developing a VTE:

Medical patients:

- significant reduction in mobility for 3 days or more (or anticipated to have significantly reduced mobility)

Surgical/trauma patients:

- hip/knee replacement
- hip fracture
- general anaesthetic and a surgical duration of over 90 minutes
- surgery of the pelvis or lower limb with a general anaesthetic and a surgical duration of over 60 minutes
- acute surgical admission with an inflammatory/intra-abdominal condition
- surgery with a significant reduction in mobility

General risk factors:

- active cancer/chemotherapy
- aged over 60
- known blood clotting disorder (e.g. thrombophilia)
- BMI over 35
- dehydration
- one or more significant medical comorbidities (e.g. heart disease; metabolic/endocrine pathologies; respiratory disease; acute infectious disease and inflammatory conditions)
- critical care admission
- use of hormone replacement therapy (HRT)
- use of the combined oral contraceptive pill
- varicose veins
- pregnant or less than 6 weeks post-partum

After a patients VTE risk has been assessed, this should be compared to their risk of bleeding to decide whether VTE prophylaxis should be offered. If indicated VTE prophylaxis should be started as soon as possible.

Types of VTE prophylaxis

Mechanical:

- Correctly fitted anti-embolism (aka compression) stockings (thigh or knee height)
- An Intermittent pneumatic compression device

Pharmacological:

- Fondaparinux sodium (SC injection)
- Low molecular weight heparin (LMWH)
    - e.g. enoxaparin
    - reduced doses should be used in patients with severe renal impairment
- Unfractionated heparin (UFH)
    - used as an alternative to LWMH in patients with chronic kidney disease

Management

In general, all medical patients deemed at risk of VTE after individual assessment are started on pharmacological VTE prophylaxis. This is providing the risk of VTE outweighs the risk of bleeding (this is often a clinical judgement) and there are no contraindications. Those at very high risk may be offered anti-embolic stockings alongside the pharmacological methods.

For surgical patients at low risk of VTE first-line treatment is anti-embolism stockings. If a patient is at high risk these stockings are used in conjunction with pharmacological prophylaxis.

Advice for patients

Pre-surgical interventions:

- Advise women to stop taking their combined oral contraceptive pill/hormone replacement therapy 4 weeks before surgery

Post-surgical interventions:

- Try to mobilise patients as soon as possible after surgery
- Ensure the patient is hydrated

Post procedure prophylaxis

For certain surgical procedures (hip and knee replacements) pharmacological VTE prophylaxis is recommended for all patients to reduce the risk of a VTE developing post-surgery. NICE make the following recommendations:

Procedure	Prophylaxis
Elective hip	LMWH for 10 days followed by aspirin (75 or 150 mg) for a further 28 days or LMWH for 28 days combined with anti-embolism stockings until discharge or Rivaroxaban
Elective knee	Aspirin (75 or 150 mg) for 14 days or LMWH for 14 days combined with anti-embolism stockings until discharge or Rivaroxaban
Fragility fractures of the pelvis, hip and proximal femur	The NICE guidance states the following (our bolding): > *Offer VTE prophylaxis for a **month** to people with fragility fractures of the pelvis, hip or proximal femur if the risk of VTE outweighs the risk of bleeding. Choose either:* > • *LMWH , starting 6-12 hours after surgery or* > • *fondaparinux sodium, starting 6 hours after surgery, providing there is low risk of bleeding.*

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 67 year old man is admitted to the Emergency Department following a deliberate overdose of an unknown drug. On assessment he is unwell and vomiting although he is maintaining an airway. Oxygen saturations are 100% on 15L/min supplemental oxygen and his respiratory rate is 32/min. His chest is clear and heart sounds are rapid. An ECG shows sinus tachycardia with a rate of 135bpm, a broad QRS complex and a QTc 520msec. His skin is flushed and hot. He complains of ringing in his ears and visual blurring as well as abdominal pain. Tympanic temperature is 37.5°C.

Blood results show:

Haemoglobin	101g/L	Sodium	131mmol/L	pH	7.11
White cells	7.8x10^9/L	Potassium	2.9mmol/L	pCO2	2.4kPa
Platelets	97x10^9/L	Urea	9.3mmol/L	pO2	78.4kPa
INR	1.3	Creatinine	198µmol/L	HCO3-	6.3mmol/L
APTT	33secs	Glucose	2.8mmol	BE	-19.7
		Chloride	94mmol/L	Lactate	2.4mmol/L

During assessment he has a generalised tonic-clonic seizure.

Which of the following drugs is he most likely to have overdosed on?

- Ibuprofen
- Isocarboxazid
- Paracetamol
- Quinine
- Venlafaxine

Submit answer

Reference ranges

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APTT	33secs	Glucose	2.8mmol	BE	-19.7
		Chloride	94mmol/L	Lactate	2.4mmol/L

During assessment he has a generalised tonic-clonic seizure.

Which of the following drugs is he most likely to have overdosed on?

Ibuprofen	4%
Isocarboxazid	4%
Paracetamol	3%
Quinine	73%
Venlafaxine	16%

Quinine toxicity (cinchonism) presents with myriad **ECG changes**, hypotension, metabolic acidosis, **hypoglycaemia** and classically tinnitus, flushing and visual disturbances. Flash pulmonary oedema may occur

Important for me Less important

Paracetamol toxicity presents with early hepatotoxicity and coagulopathy, and later renal failure and encephalopathy which are features lacking in this clinical scenario.

Ibuprofen, a non-steroidal anti-inflammatory, is relatively safe in overdose although massive ingestion may cause gastrointestinal ulceration and haemorrhage as well as electrolyte imbalances, coagulopathy and prominent cerebellar signs. This is not commented upon in the clinical vignette.

Venlafaxine is a selective serotonin and noradrenaline reuptake inhibitor and presents with symptoms of serotonin and sympathetic overdrive in poisoning, including agitation, sweating, hyperpyrexia, clonus, tachycardia and hypertension. ECG abnormalities similar to the above may occur, as may hypoglycaemia but venlafaxine is not the most likely agent.

Isocarboxazid is a monoamine oxidase inhibitor and in overdose may also cause symptoms of the serotonin syndrome although sympathomimetic features tend to predominate, including hyperglycaemia.

Discuss (4) Improve

Next question

Quinine toxicity (cinchonism)

Quinine is a remarkably toxic drug; something which is not so readily acknowledged. It is used as an antimalarial drug and also as a prophylactic agent against leg cramps, although both uses are increasingly falling from vogue due to the availability of better, safer agents. Quinine toxicity, known as cinchonism, may be fatal, usually by cardiac arrhythmia or flash pulmonary oedema in the short term, although incipient renal failure may be fatal more long-term.

Cardiac arrhythmia is a common finding in cinchonism due to blockade of sodium and potassium channels prolonging QRS and QT intervals respectively and these rhythms may degenerate into ventricular tachyarrhythmias or fibrillation causing death. **Hypoglycaemia** is also a common finding in cinchonism since quinine stimulates pancreatic insulin secretion and this should be corrected rapidly if present. Unlike in the above case, flash pulmonary oedema may develop causing hypoxia and necessitating positive pressure ventilation. Classical hallmarks of cinchonism are tinnitus, visual blurring, flushed and dry skin and abdominal pain.

Clinically, quinine toxicity is difficult to distinguish from aspirin poisoning and so measurement of serum salicylate levels is important when this clinical picture is seen. In terms of management however, whereas aspirin can be cleared from overdose victims by haemofiltration, quinine cannot be extracted easily by extracorporeal methods. Central nervous symptoms such as tinnitus,

deafness and visual defects which may occur with aspirin are usually transient whereas quinine leaves permanent neural damage, if the patient survives.

Management of quinine poisoning is largely supportive with fluids, inotropes and bicarbonate as needed as well as positive pressure ventilation for pulmonary oedema.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 28-year-old female with a history of cerebral palsy is admitted for an elective intrathecal pump refill. The procedure unfortunately was unsuccessful and the pump could not be re-sited. Later on in the evening the patient complains of severe pain in her lower limbs and feels generally unwell.

Blood results are as follows:

Hb	145 g/l	Na^+	138 mmol/l
Platelets	525 * 10^9/l	K^+	5.6 mmol/l
WBC	14.1 * 10^9/l	Urea	12.8 mmol/l
Neuts	8.4 * 10^9/l	Creatinine	156 µmol/l
Creatine kinase	8445 Units/litre (normal range 24-170)	CRP	44 mg/l

What drug is the patient withdrawing from?

- Lidocaine
- Baclofen
- Pethidine
- Clonidine
- Ketorolac

Submit answer

Reference ranges

Score: **0%**

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Neuts	8.4 * 10^9/l	Creatinine	156 µmol/l
Creatine kinase	8445 Units/litre (normal range 24-170)	CRP	44 mg/l

What drug is the patient withdrawing from?

Lidocaine	11%
Baclofen	59%
Pethidine	12%
Clonidine	9%
Ketorolac	8%

Intrathecal baclofen withdrawal syndrome is associated with severe spasticity, rhabdomyolysis, acute renal failure and multisystem organ failure

Important for me Less important

Intrathecal baclofen can be used to treat spasticity. It can be administered continuously via a pump. It is believed to work by agonising GABA receptors.

Discontinuation of baclofen can be associated with a withdrawal syndrome which resembles benzodiazepine withdrawal and alcohol withdrawal. Intrathecal baclofen withdrawal syndrome is associated with severe spasticity, rhabdomyolysis, acute renal failure and multisystem organ failure.

Drugs acting on common receptors

The table below lists the common receptors and gives examples of both agonists and antagonists. It is not comprehensive and is intended to give examples of each.

Receptor	Agonist	Antagonist
Alpha	Alpha-1: Decongestants (e.g. phenylephrine/oxymetazoline) Alpha-2: Glaucoma (e.g. topical brimonidine)	Benign prostatic hyperplasia (e.g. tamsulosin) Hypertension (e.g. doxazosin)
Beta-1	Inotropes (e.g. dobutamine)	Non-selective & selective beta-blockers (e.g. atenolol, bisoprolol)
Beta-2	Bronchodilators (e.g. salbutamol)	Non-selective beta-blockers (e.g. propranolol, labetalol)
Dopamine	Parkinson's disease (e.g. ropinirole) Prolactinoma	Schizophrenia (antipsychotics e.g. haloperidol) Anti-emetics (e.g. metoclopramide/domperidone)
GABA	Benzodiazepines Baclofen	Flumazenil
Histamine-1		Antihistamines (e.g. loratadine)
Histamine-2		Antacids (e.g. ranitidine)
Muscarinic	Glaucoma (e.g. pilocarpine)	Atropine (e.g. for bradycardia) Bronchodilator (e.g. ipratropium bromide, tiotropium) Urge incontinence (e.g. oxybutynin)
Nicotinic	Nicotine Varenicline (used for smoking	Non-depolarising muscle relaxants (e.g. atracurium)

Receptor	Agonist	Antagonist
	cessation) Depolarising muscle relaxant (e.g. suxamethonium)	
Oxytocin	Inducing labour (e.g. Syntocinon)	Tocolysis (e.g. atosiban)
Serotonin	Triptans (for acute migraine, e.g. zolmitriptan)	Anti-emetics (e.g. ondansetron)

Next question

Textbooks

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Score: **15.1%**

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A 76-year-old man presented with shortness of breath to the emergency department. On the basis of a recent knee replacement operation and unremarkable chest x-ray a CT pulmonary angiogram was performed and demonstrated a segmental pulmonary embolus with evidence of mild right heart strain. The patient was haemodynamically stable and required only minimal supplemental oxygen therapy.

The patient had known chronic kidney disease stage IV secondary to type 2 diabetes and hypertension. Treatment was therefore initiated with an intravenous unfractionated heparin infusion.

The patient's condition was stable over the following week with warfarin loading cautiously started at day 6 of admission. Routine blood tests at this point indicated a new abnormality in full blood count leading to further investigations as detailed below.

Haemoglobin	14.5 g / dL
White blood cells	8.6 x 10>3 / microlitre
Neutrophils	4.5 x 10>3 / microlitre
Lymphocytes	2.1 x 10>3 / microlitre
Platelets	67 x 10>3 / microlitre
Mean cell volume	85 fL
Mean cell haemoglobin	30.1 pg
B12	252 pmol / L
Folate	20 nmol / L

Heparin induced thrombocytopenia antibodies: positive (high titre)

Following cessation of IV heparin infusion, what is the appropriate management of the patient's thrombocytopenia?

- Bivalirudin
- Enoxaparin
- Warfarin

Tirofiban

Platelet transfusion

Submit answer

Reference ranges

Score: **0%**

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2 -
3 -
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B12	252 pmol / L
Folate	20 nmol / L

Heparin induced thrombocytopenia antibodies: positive (high titre)

Following cessation of IV heparin infusion, what is the appropriate management of the patient's thrombocytopenia?

Bivalirudin	75%
Enoxaparin	5%
Warfarin	5%

Tirofiban	13%
Platelet transfusion	2%

This patient has a confirmed diagnosis of heparin induced thrombocytopenia (HIT) based on the suggestive clinical picture and positive HIT antibody screen. Decision tools such as the Warkentin probability scale can be useful by providing a pre-test probability score of HIT based on the clinical picture. In this case, the timing of thrombocytopenia with onset between 5-10 days after initial exposure to heparin is highly suggestive.

In suspected or confirmed cases of HIT, heparin anticoagulants must be withheld immediately. Treatment with a non-heparin anticoagulant such as bivalirudin should be started to reduce the risk of HIT-related thrombosis.

Vitamin K antagonists such as warfarin such be withheld or reversed as they do not prevent HIT-associated thrombosis and increase the risk of venous gangrene. Platelet transfusion would not be indicated in this patient.

Linkins L-A. Heparin induced thrombocytopenia. BMJ 2014;349:7566.

Discuss (3) Improve

Next question

Heparin

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	Low molecular weight heparin (LMWH)
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

AI + Q 123

Next question >

Textbooks

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Links

BNF

3 2

Heparin

British Society for Haematology

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2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

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A 24-year-old man is brought to the emergency department having developed acute confusion and fever. The family tell you he was well on going to bed last night then become poorly over several hours this morning.

He has a background of depression for which he takes sertraline. His mother tells you he has recently been trying some over-the-counter remedies for his depression and has also been complaining of lower back pain, for which he has started new painkillers. A set of observations are available.

Respiratory rate 30/min
Oxygen saturation 97% on air
Heart rate 120/min
Blood pressure 184/106mmHg
Temperature 39.1ºC

On examination, you find him to be agitated and disorientated. Neurological assessment reveals generalised rigidity and brisk reflexes. His pupils are dilated and you note marked myoclonus at both ankles. An ECG shows sinus tachycardia with a normal QRS duration.

What treatment is indicated?

- Cyproheptadine
- Dantrolene
- Glucagon
- N-acetylcysteine
- Sodium bicarbonate

Submit answer

Reference ranges

Score: **0%**

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On examination, you find him to be agitated and disorientated. Neurological assessment reveals generalised rigidity and brisk reflexes. His pupils are dilated and you note marked myoclonus at both ankles. An ECG shows sinus tachycardia with a normal QRS duration.

What treatment is indicated?

Cyproheptadine	74%
Dantrolene	20%
Glucagon	1%
N-acetylcysteine	1%
Sodium bicarbonate	5%

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

Important for me Less important

The correct answer is **cyproheptadine**. This is a 5-HT2 receptor antagonist used in the treatment of serotonin syndrome. Key features include rigidity, tachycardia, hyperpyrexia and hypertension. In this case, the rapid onset, dilated pupils and brisk reflexes support this as the diagnosis. The over-the-counter remedy and new painkiller may represent St. John's wort or tramadol, both of which may interact with SSRIs such as sertraline to induce this syndrome.

Dantrolene is incorrect. This is the treatment for neuroleptic malignant syndrome. This syndrome is associated with antipsychotics rather than anti-depressants. It also generally is of slower onset than serotonin syndrome and presents with normal pupils and diminished reflexes.

Glucagon is incorrect. This is a treatment used in the management of beta-blocker overdose. There is no suggestion this patient has ingested these in the case described. The presentation is also not in keeping with beta-blocker toxicity, particularly tachycardia.

N-acetylcysteine is incorrect. This is the antidote for paracetamol overdose. Although this may be the painkiller he is taking for his back pain, it would not account for the clinical features described. Paracetamol overdose generally causes hepatotoxicity.

Sodium bicarbonate is incorrect. This can be used in the management of tricyclic antidepressant overdose. The case does not describe ingestion of these, and significant toxicity is associated with a widening of the QRS complex leading to ventricular arrhythmias. In this case, we are told the QRS is normal, which would go against significant TCA toxicity.

Discuss (2) | Improve

Next question >

Serotonin syndrome

Causes

- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

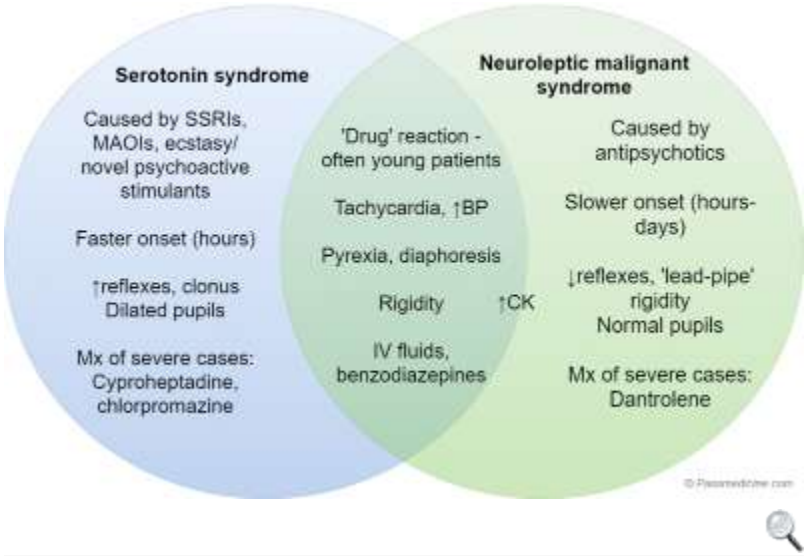
Features

- neuromuscular excitation

    - hyperreflexia
    - myoclonus
    - rigidity
- autonomic nervous system excitation
    - hyperthermia
    - sweating
- altered mental state
    - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question

Textbooks

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Links

Medsafe 1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

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A 17 year old girl is brought to the Emergency Department following a prolonged seizure lasting seven minutes. She is known to suffer from epilepsy and has been stable on lamotrigine for eight years, having on average only one generalised tonic clonic seizure every eight months. Her mother describes an increase in her seizure frequency over the last four weeks; she has has three generalised tonic-clonic seizures during this period, each lasting around three minutes. There have been no changes to stress levels, sleep pattern or diet. She confirms she has however started a new medication for dysmenorrhoea. Which is the most likely to explain this increase in frequency?

- Medroxyprogesterone (Depo-Provera)
- Mefenamic acid
- Ibuprofen
- Aspirin
- Naproxen

Submit answer

Reference ranges

Score: **0%**

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Medroxyprogesterone (Depo-Provera)	42%
Mefenamic acid	44%
Ibuprofen	5%
Aspirin	4%
Naproxen	6%

Taking a full medication history is an essential aspect of any history; it can be particularly important in epilepsy, not only to gauge medication compliance and anti-epileptic therapy, but to determine any precipitating factors.

Analgesics that are known to induce seizures or worsen seizure control include fentanyl, mefenamic acid, and tramadol (among others). Other drugs that are known to induce seizures include amitriptyline, aminophylline, isotretinoin and haloperidol.

It is important to be aware of the interaction between contraception and antiepileptic medication; contraceptive failure, teratogenicity and reduced seizure control are potential effects.

P450 enzyme inducers will decrease the drug level and therefore increase the failure rate of oestrogen and progesterone containing contraceptives.

- Carbamazepine
- Oxcarbazepine
- Phenytoin
- Phenobarbitone
- Primidone
- Topiramate

Lamotrigine is not a P450 inducer. Studies have shown a slight decrease in levonorgestrel levels, although the clinical significance is undetermined. Levels of lamotrigine are reduced by oral contraceptives containing ethinylestradiol, increased seizure frequency may therefore occur. There is no evidence that progestogen-only methods affect lamotrigine levels.

Sources:

- BNF:4.8.1 control of the epilepsies
- Centers for Disease Control and Prevention. Morbidity and Mortality Weekly Report (MMWR). U.S. Medical Eligibility Criteria for Contraceptive Use, (http://www.cdc.gov/mmwr/preview/mmwrhtml/rr59e0528a1.htm)
- Faculty of sexual and reproductive healthcare clinical effectiveness unit; antiepileptic drugs and contraception. FSRH 2010

Discuss (5) | Improve

Next question

Prescribing in patients with epilepsy

The following drugs may worsen seizure control in patients with epilepsy:

- alcohol, cocaine, amphetamines
- ciprofloxacin, levofloxacin
- aminophylline, theophylline
- bupropion
- methylphenidate (used in ADHD)
- mefenamic acid

Some medications such as benzodiazepines, baclofen and hydroxyzine may provoke seizures whilst they are being withdrawn.

Other medications may worsen seizure control by interfering with the metabolism of anti-epileptic drugs (i.e. P450 inducers/inhibitors).

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 34-year-old man is seen in the emergency department at 5 am on Saturday morning in a state of extreme agitation. He is alert from the end of the bed, tremulous and diaphoretic. His respiratory rate is 26 breaths per minute, oxygen saturation 97% on air, heart rate 132 beats per minute, blood pressure 185/103mmHg and temperature 38.2ºC. There is bilateral air entry on auscultation of the chest with vesicular breath sounds. His peripheries are clammy, hot to touch and his pulse is fast and bounding. His heart sounds are normal with no added murmurs. His JVP is not raised. He is talking to you but is confused. Pupils are equal and reactive to light but markedly dilated. There is no lateralising neurology.

The nurse cannulates the patient and takes blood.

The initial investigation results are as follows:

ECG	sinus tachycardia, 1mm T wave inversions in V2-V6
Urine dipstick	+ protein +++ haemoglobin

Venous blood gas:

pH	7.32	7.31-7.41
pO_2	5.35 kPa	4.0-5.3 kPa
pCO_2	4.73 kPa	5.5-6.8 kPa
Lac	3.2 mmol/L	<2.0 mmol/L
Glucose	6.4 mmol/L	4.0-7.8 mmol/L
HCO3	24 mmol/L	23-29 mmol/L
BE	-3	-2 - +2

What would be the next most appropriate management step?

- Aspirin, clopidogrel, fondaparinux and sublingual glycerine trinitrate
- CT aortogram
- IV benzodiazepines

- IV labetalol
- IV 8.4% sodium bicarbonate

Submit answer

Reference ranges

Score: **0%**

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HCO3	24 mmol/L	23-29 mmol/L
BE	-3	-2 - +2

What would be the next most appropriate management step?

Aspirin, clopidogrel, fondaparinux and sublingual glycerine trinitrate	6%
CT aortogram	3%
IV benzodiazepines	72%

IV labetalol 7%

IV 8.4% sodium bicarbonate 13%

Treatment of cocaine toxicity is largely supportive with judicious usage of benzodiazepines

Important for me Less important

This man displays signs of cocaine toxicity with dilated pupils, hypertension, tachycardia, anxiety, likely myoglobinuria secondary to rhabdomyolysis and myocardial strain. The management would involve IV benzodiazepines. Nitroglycerine can also be useful in the face of coronary vasospasm.

It is important to bear in mind that β-blockers are contraindicated in cocaine toxicity due to the risks of the unopposed α-agonist activity. For this reason, IV labetalol would not be the next most appropriate management step.

Aspirin, clopidogrel, fondaparinux and sublingual glycerine trinitrate would be the first line if you suspected a non-ST elevated myocardial infarction. In this case, the main concern is cocaine toxicity which is causing signs of rate-dependent ischemia on the ECG. First line management would be benzodiazepines which would help to resolve this issue.

A CT aortogram would be useful if you suspected aortic dissection. There is no strong evidence that this is occurring and cocaine toxicity is the more likely diagnosis in this case.

IV 8.4% sodium bicarbonate is useful for overdoses of tricyclic antidepressant but it is not the next most appropriate management step in the case of cocaine toxicity.

Discuss (6) Improve

Next question

Cocaine

Cocaine is an alkaloid derived from the coca plant. It is widely used as a recreational stimulant. The price of cocaine has fallen sharply in the past decade resulting in cocaine toxicity becoming a much more frequent clinical problem. This increase has made cocaine a favourite topic of question writers.

Mechanism of action

- cocaine blocks the uptake of dopamine, noradrenaline and serotonin

Adverse effects

- cardiovascular
  - coronary artery spasm → myocardial ischaemia/infarction
  - both tachycardia and bradycardia may occur
  - hypertension
  - QRS widening and QT prolongation
  - aortic dissection
- neurological
  - seizures
  - mydriasis
  - hypertonia
  - hyperreflexia
- psychiatric effects
  - agitation
  - psychosis
  - hallucinations
- others
  - ischaemic colitis is recognised in patients following cocaine ingestion. This should be considered if patients complain of abdominal pain or rectal bleeding
  - hyperthermia
  - metabolic acidosis
  - rhabdomyolysis

Management of cocaine toxicity

- in general, benzodiazepines are generally first-line for most cocaine-related problems
- chest pain:
  - benzodiazepines + glyceryl trinitrate
  - if myocardial infarction develops then primary percutaneous coronary intervention
- hypertension: benzodiazepines + sodium nitroprusside
- the use of beta-blockers in cocaine-induced cardiovascular problems is a controversial issue
  - the American Heart Association issued a statement in 2008 warning against the use of beta-blockers (due to the risk of unopposed alpha-mediated coronary vasospasm) but many cardiologists since have questioned whether this is valid
  - if a reasonable alternative is given in an exam it is probably wise to choose it

AI + Q 123

Next question ›

B I A T↕

Textbooks

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Media

Cocaine Dependence

Osmosis - YouTube 4 2

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A 55-year-old man presents to the Emergency Department after taking an overdose. He can not tell you which of his regular tablets he has taken an overdose of. His past medical history includes Crohn's disease, gastroesophageal reflux disease, hypertension, gout, depression and paroxysmal atrial fibrillation.

He is complaining of chest pain and shortness of breath. Observations - respiratory rate 26/min, saturations 94% on air, heart rate 50/min, blood pressure 75/40 mmHg, temperature 37.2ºC.

His ECG shows atrial fibrillation with a broad QRS.

Which drug is the most likely culprit?

- Flecainide
- Amitriptyline
- Allopurinol
- Azathioprine
- Diltiazem

Submit answer

Reference ranges

Score: **0%**

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He is complaining of chest pain and shortness of breath. Observations - respiratory rate 26/min, saturations 94% on air, heart rate 50/min, blood pressure 75/40 mmHg, temperature 37.2ºC.

His ECG shows atrial fibrillation with a broad QRS.

Which drug is the most likely culprit?

Flecainide	24%
Amitriptyline	57%
Allopurinol	1%
Azathioprine	1%
Diltiazem	16%

Flecainide is the most likely culprit here due to the chest pain, signs of heart failure and prolonged QRS. Diltiazem would be also be possible, but in overdose would likely show a more marked bradycardia with heart block.

Flecainide is a Vaughan Williams Class Ic anti-arrhythmic drug, associated with PR, QRS, and QTc prolongation on the electrocardiogram and development of life-threatening cardiac toxicity in overdose.

The cornerstone of treatment is fluid resuscitation and the administration of magnesium and sodium bicarbonate. Intravenous fat emulsion has also been trialed with some success.

Note - flecainide should NOT be used in patients who have a structurally abnormal heart due to the risk of causing arrhythmias, including torsades de pointes.

Flecainide

Flecainide is a Vaughan Williams class 1c antiarrhythmic. It slows conduction of the action potential by acting as a potent sodium channel blocker (specifically the Nav1.5 sodium channels). This may be reflected by widening of the QRS complex and prolongation of the PR interval.

The Cardiac Arrhythmia Suppression Trial (CAST, 1989) investigated the use of agents to treat asymptomatic or mildly symptomatic premature ventricular complexes (PVCs) post myocardial infarction. The hypothesis was that this would reduce deaths from ventricular arrhythmias. Flecainide was actually shown to increase mortality post-myocardial infarction and is, therefore, contraindicated in this situation.

Indications

- atrial fibrillation
- SVT associated with accessory pathway e.g. Wolf-Parkinson-White syndrome

Contraindications

- post myocardial infarction
- structural heart disease: e.g. heart failure
- sinus node dysfunction; second-degree or greater AV block
- atrial flutter

Adverse effects

- negatively inotropic
- bradycardia
- proarrhythmic
- oral paraesthesia
- visual disturbances

Textbooks

High-yield textbook

Extended textbook

Links

BNF

Flecainide

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Suggest link

Report broken link

Score: **15.1%**

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A 27 year old well-nourished male electroplater is admitted to hospital with abdominal pain and neurological symptoms. He tells you that he has had worsening symptoms for the past three months with severe central abdominal pain, diarrhoea, vomiting and painful burning sensation in both feet. He also describes increasing clumsiness with loss of manual dexterity and tripping over his feet frequently. He describes dimming of his vision over the past two weeks with a sepia tinge. On examination, there is mild voluntary guarding of the abdomen but no discrete masses and no organomegaly. The cardiorespiratory examination is normal. Examination of the neurological system reveals distal weakness with MRC power grade 4/5 in all limbs but normal proximal power. There is a mild tremor present with his arms outstretched and trunk ataxia. Sensory examination discloses painful paraesthesia in the hands and feet with hyperalgesia. There is a loss of proprioception. The cranial nerves are notable in that a left sixth nerve palsy is present with accompanying diplopia. Visual acuity is reduced to 6/30 bilaterally and eye movements are painful. He has a bilateral ptosis. He is noted to have minimal body hair, including hairless arms and legs, lateral third of the eyebrow and temporal and crown baldness which he tells you occurred in the past month. He also has scaling of the palms and soles, tender glossitis and transverse white lines on all his nails. He is cyclothymic during assessment.

What is the most appropriate management for this patient?

- High dose IV hydrocortisone 200mg three times daily
- High dose IV vitamin B complex (Pabrinex I+II) and folinic acid
- Intravenous immunoglobulins
- Oral Prussian Blue 10g twice daily
- Procaine penicillin (G) 2 million unit daily IM with oral probenicid

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate management for this patient?

High dose IV hydrocortisone 200mg three times daily	5%
High dose IV vitamin B complex (Pabrinex I+II) and folinic acid	35%
Intravenous immunoglobulins	6%
Oral Prussian Blue 10g twice daily	45%
Procaine penicillin (G) 2 million unit daily IM with oral probenicid	9%

Thallium poisoning is a rare cause of painful polyneuropathy, mood change and alopecia. Treatment is chelation therapy with oral Prussian Blue

Important for me Less important

The above clinical descriptor is classical for thallium poisoning. The triad of fluctuating mood, with or without confusion, painful distal paraesthesia and alopecia is virtually pathognomonic of toxicity with this heavy metal.

Thallium is a highly toxic metal; in its univalent Tl+ ion it is readily soluble in water and easily enters the body across the skin or gastrointestinal tract using potassium influx channels. Thallium fumes may also be generated on heating this metal and these may be inhaled when using it during many electroplating manufacturing processes. In the UK, the Health and Safety Executive stipulates that worker occupational exposure limits to thallium in any form must not exceed 0.1mg/m3 in an 8 hour period without adequate ventilation due to the health risks.

Thallium avidly binds to sulphur ligands within cellular proteins, disrupting many intracellular metabolic processes. In massive exposure, death from neuromuscular respiratory failure is rapid, but in more prolonged exposure insidious symptoms of neuropathy, abdominal pain, gastrointestinal bleeding, optic neuritis, confusion and weakness occur. Skin changes such as palm and sole scaling, stomatitis, glossitis, keratitis, eczema and classical alopecia and Mee's lines on the nails typically occur within two weeks of continual exposure. Treatment is with Prussian Blue which is administered orally at a dose of 250-300mg/kg/day (approximately 10g twice daily for an adult) and this complexes with thallium and is excreted in the faeces.
Thallium poisoning is often diagnosed late or not at all due to a low index of suspicion; indeed it is often misdiagnosed as Guillain-Barre syndrome due to the glove and stocking distribution of sensory symptoms and patients may receive intravenous steroids or immunoglobulins unnecessarily. In Guillain-Barre syndrome (GBS), classically there is ascending paralysis that is not seen in thallium poisoning and typically sensory symptoms in GBS are of numbness not hyperalgesia.

Thallium poisoning has also been mistaken for neurosyphilis and severe vitamin B deficiency. While neurosyphilis shares symptoms of confusion, fluctuating mood and palmar lesions as well as neuropathy, abdominal symptoms are usually not present and the diagnostic alopecia is absent in syphilis. Similarly, conditions such as Pellagra (niacin deficiency) and beriberi (thiamine deficiency) may share similar features with thallium poisoning such as alopecia, skin changes and mood disturbances, a painful neuropathy is less common; indeed sensory loss is more common in the extremities. Also in these conditions, the prime pathological aetiology is dietary deficiency and the history gives no suggestion this is an issue in this case.

👍 👎 💬 Discuss (14) Improve

Next question ›

Thallium poisoning ★

Features

- painful polyneuropathy
- mood change
- alopecia

Treatment is chelation therapy with oral Prussian Blue.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 72-year-old male was admitted with 72 hours of progressive ascending motor weakness associated with palpitation and back pain. A diagnosis of Guillain-Barre syndrome was made and he was started on intravenous immunoglobulin (IVIg) with intensive monitoring on the neurology ward. You are asked to review the bloods of the patient on the fourth of five days of IVIg treatment. You note his sodium to have changes as follows: Na 145 → 141 → 134 → 130 → 126. The patient reports 'feeling better after the drip', denies any cough, shortness of breath, diarrhoea, nausea or vomiting. On examination, he appears comfortable in bed, JVP 2/3 cm above the angle of Louis, has warm peripheries and capillary refill time of 2 seconds. His chest is clear on auscultation, cardiovascular examination unremarkable and neurological examination unchanged from admission. What is your management plan?

- No action
- Intravenous fluid
- Fluid restrict to 1.5 litres
- Contact endocrinology team regarding possible syndrome of inappropriate anti-diuretic hormone (SIADH)
- Contact intensive care unit regarding escalation and intubation

Submit answer

Reference ranges

Score: **0%**

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No action	26%
Intravenous fluid	6%
Fluid restrict to 1.5 litres	44%
Contact endocrinology team regarding possible syndrome of inappropriate anti-diuretic hormone (SIADH)	19%
Contact intensive care unit regarding escalation and intubation	4%

The patient has received four days of intravenous immunoglobulin (IVIg), which is a protein and hence likely to distort the measurement of sodium, producing a pseudohyponatraemia. This is also observed in patients with high serum concentrations of triglycerides, cholesterol and in those with high serum protein such as myeloma patients. This patient is clinically euvolaemic. No action is required.

Discuss (7) | Improve

Next question

Immunoglobulins: therapeutics

The Department of Health issued guidelines on the use of intravenous immunoglobulins in May 2008

Uses

- primary and secondary immunodeficiency
- idiopathic thrombocytopenic purpura
- myasthenia gravis
- Guillain-Barre syndrome
- Kawasaki disease
- toxic epidermal necrolysis
- pneumonitis induced by CMV following transplantation
- low serum IgG levels following haematopoietic stem cell transplant for malignancy
- dermatomyositis
- chronic inflammatory demyelinating polyradiculopathy

Basics

- formed from large pool of donors (e.g. 5,000)
- IgG molecules with a subclass distribution similar to that of normal blood
- half-life of 3 weeks

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Department of Health 1 1

2011 Guidelines on the clinical use of immunoglobulins

Suggest link Report broken link

Score: **15.1%**

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A 63-year-old man is commenced on anti-tuberculosis therapy, after presenting to the hospital with a history of several months of weight loss, shortness of breath and a productive cough which was sometimes accompanied by haemoptysis.

A chest x-ray had revealed apical consolidation and sputum was positive for acid-fast bacilli, following which a diagnosis of tuberculosis was then made. His only past medical history includes a diagnosis of asthma for which he takes PRN salbutamol and Seretide (25 micrograms of salmeterol and 125 micrograms of fluticasone propionate) inhaler twice daily.

A week after being diagnosed, he is admitted to the acute medical unit with shortness of breath and bilateral expiratory wheeze. His observations reveal a temperature of 37.1ºC, respiratory rate of 27 breaths per minute and oxygen saturations of 94% on room air. Oxygen is commenced via a face mask. A chest x-ray reveals the same apical consolidation as before, with no new features.

What is the most appropriate next step in her treatment?

- Continue the tuberculosis medication at current dose and increase the steroid dose
- Continue the tuberculosis medication at current dose and decrease the steroid dose
- Reduce the tuberculosis medication and increase the steroid dose
- Stop the tuberculosis medication and increase the steroid dose
- Continue the tuberculosis medication at current dose and keep the steroid dose the same

Submit answer

Reference ranges

Score: **0%**

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What is the most appropriate next step in her treatment?

Continue the tuberculosis medication at current dose and increase the steroid dose	72%
Continue the tuberculosis medication at current dose and decrease the steroid dose	10%
Reduce the tuberculosis medication and increase the steroid dose	4%
Stop the tuberculosis medication and increase the steroid dose	3%
Continue the tuberculosis medication at current dose and keep the steroid dose the same	11%

Rifampicin increases the metabolism of corticosteroids, thus patients who are on long-term steroids (as in this case) should receive a higher dose of corticosteroids when starting tuberculosis therapy.

Discuss (4) | Improve

Next question

Tuberculosis: drug side-effects and mechanism of action

Rifampicin

- mechanism of action: inhibits bacterial DNA dependent RNA polymerase preventing transcription of DNA into mRNA
- potent liver enzyme inducer
- hepatitis, orange secretions
- flu-like symptoms

Isoniazid

- mechanism of action: inhibits mycolic acid synthesis
- peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
- hepatitis, agranulocytosis
- liver enzyme inhibitor

Pyrazinamide

- mechanism of action: converted by pyrazinamidase into pyrazinoic acid which in turn inhibits fatty acid synthase (FAS) I
- hyperuricaemia causing gout
- arthralgia, myalgia
- hepatitis

Ethambutol

- mechanism of action: inhibits the enzyme arabinosyl transferase which polymerizes arabinose into arabinan
- optic neuritis: check visual acuity before and during treatment
- dose needs adjusting in patients with renal impairment

AI + Q 123

Next question >

B I A T

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2016 Tuberculosis guidelines

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Suggest link

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Media

Tuberculosis

Osmosis - YouTube 0 0

Report broken media

Score: **15.1%**

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A 27-year-old man presents to his GP with generalised itchiness. This has been ongoing for some weeks and he has tried a number of creams over the counter. The GP provided a steroid cream and chlorphenamine tablets which helped initially but he is now finding he cannot sleep due to the itching. He has become tired and has been late to work as a fitness instructor several times. He denies alcohol use and is an ex smoker. His GP decides to perform some blood tests.

On examination, this well built gentleman looks well with scleral icterus present. There are numerous scratch marks across his torso and he has mild gynaecomastia. His abdomen is soft with no palpable masses. There are no spider naevi. His ankles are soft with no oedema. He has no rashes.

He mother has ulcerative colitis with liver complications and he has no past medical history.

Hb	180 g/l	Na^+	145 mmol/l	Bilirubin	45 µmol/l
Platelets	459 * 10^9/l	K^+	3.0 mmol/l	ALP	189u/l
WBC	9.8 * 10^9/l	Urea	4.5 mmol/l	ALT	61 u/l
Neuts	7.2 * 10^9/l	Creatinine	95 µmol/l	γGT	213 u/l
Lymphs	1.2 * 10^9/l	Albumin	45 g/l		
Anti smooth muscle antibody	negative	Anti liver kidney antibody	negative		

What is the likeliest cause of his deranged bloods?

- Primary biliary cirrhosis
- Alcoholic fatty liver disease
- Primary sclerosing cholangitis
- Anabolic steroid
- Wilson's Disease

Submit answer

Reference ranges

Score: **0%**

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Lymphs	1.2 * 10^9/l	Albumin	45 g/l		
Anti smooth muscle antibody	negative	Anti liver kidney antibody	negative		

What is the likeliest cause of his deranged bloods?

- Primary biliary cirrhosis — 9%
- Alcoholic fatty liver disease — 1%
- Primary sclerosing cholangitis — 24%
- Anabolic steroid — 65%
- Wilson's Disease — 1%

Anabolic steroids are a cause of cholestasis

Important for me Less important

This gentleman presents with a symptomatic LFT rise. The ALP is raised out of proportion to the ALT making this a cholestatic picture which rules out AFLD and Wilson's disease. He has a family history for autoimmune disease but his antibodies are negative making PBC less likely. He is a well built gentleman and works in the fitness industry. Anabolic steroid use is highly prevalent especially with easy access through online sales. This gives an isolated cholestasis but can also give a polycythaemia through erythropoeisis and causes hypernatraemia and hypokalaemia. This should be discussed with the gentleman.

Discuss (8) | Improve

Next question

Anabolic steroid use

Anabolic steroid use is associated with several serious long-term health consequences. Cardiac morbidity and mortality are increased by anabolic steroid use, although the precise mechanism of this effect is unclear. Hepatic side effects also occur secondary to chronic vascular injury: these include hepatocellular carcinoma and hepatic adenoma. Psychiatric illness is also commonly co-morbid with anabolic steroid use. Additionally, users who inject anabolic steroids have an increased risk of blood-borne viruses if needles are shared between individuals.

Due to the above concerns, patients should be strongly counselled to stop using anabolic steroids. There is no requirement for tapering of doses. Many of the above blood test abnormalities normalise once anabolic steroid consumption ceases. The expert recommendation is for lifelong monitoring for potential complications, initially annually with frequency reducing once blood markers normalise and in the absence of apparent adverse effects.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 29-year-old man is brought to the emergency department after the sudden onset of severe left-sided chest pain.

His observations are taken as follows: temperature 37.2°C, blood pressure 165/92 mmHg, pulse 129/min and regular, and respiratory rate 20/min. On examination, he is anxious, diaphoretic and agitated. His nasal mucosa is atrophic, and both pupils are dilated. On auscultation, heart sounds are regular with no murmurs or rubs, and the lungs are clear.

An initial ECG shows ST-segment elevation in leads V4-V6. A chest x-ray is normal.

His laboratory studies show:

		Reference range
Haemoglobin	165 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	8.5 * 10^9/l	4.0-11.0 * 10^9/L
Platelets	310 * 10^9/l	150-400 * 10^9/L
Sodium	139 mmol/L	135-145 mmol/L
Potassium	4.5 mmol/L	3.5-5.0 mmol/L
Urea	3.5 mmol/L	2.0-7 mmol/L
Creatinine	94 umol/L	55-120 umol/L
Troponin T	32 ng/L	<14 ng/L

Which of the following treatment options must be avoided in this patient?

- Aspirin
- Diazepam
- Diltiazem
- Labetalol
- Nitroglycerin

Submit answer

Reference ranges

Score: **0%**

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A 29-year-old man is brought to the emergency department after the sudden onset of severe left-sided chest pain.

His observations are taken as follows: temperature 37.2°C, blood pressure 165/92 mmHg, pulse 129/min and regular, and respiratory rate 20/min. On examination, he is anxious, diaphoretic and agitated. His nasal mucosa is atrophic, and both pupils are dilated. On auscultation, heart sounds are regular with no murmurs or rubs, and the lungs are clear.

An initial ECG shows ST-segment elevation in leads V4-V6. A chest x-ray is normal.

His laboratory studies show:

		Reference range
Haemoglobin	165 g/L	Men: 135-180 g/L Women: 115-160 g/L
White blood cells	8.5 * 10^9/l	4.0-11.0 * 10^9/L
Platelets	310 * 10^9/l	150-400 * 10^9/L
Sodium	139 mmol/L	135-145 mmol/L
Potassium	4.5 mmol/L	3.5-5.0 mmol/L
Urea	3.5 mmol/L	2.0-7 mmol/L
Creatinine	94 umol/L	55-120 umol/L
Troponin T	32 ng/L	<14 ng/L

Which of the following treatment options must be avoided in this patient?

Aspirin	5%
Diazepam	10%
Diltiazem	17%
Labetalol	55%
Nitroglycerin	14%

Cocaine toxicity - avoid beta-blockers

Important for me Less important

This patient is presenting with acute cocaine toxicity complicated by myocardial ischaemia. Hints pointing towards cocaine toxicity are excessive sympathetic stimulation, including tachycardia, dilated pupils and sweating. Cocaine toxicity can cause coronary artery vasospasm, giving rise to myocardial ischaemia. Troponin T levels are raised in myocardial ischaemia. The treatment of myocardial ischaemia in patients with and without cocaine intoxication is similar other than one important distinction: **patients with cocaine intoxication are not given beta-blockers**. The rationale behind these guidelines is that beta-blockage can lead to unopposed alpha-adrenergic stimulation and worsening coronary vasoconstriction.

Aspirin is routinely used to treat patients with myocardial infarction, especially those with cocaine intoxication. Cocaine can induce thrombus formation by activating platelets, stimulating their aggregation, and increasing thromboxane production. Patients with aortic dissection, another common complication of cocaine intoxication, should not be given aspirin. This patient's chest x-ray is normal, and his presentation does not point towards aortic dissection, making aspirin a recommended drug in this case. Aortic dissection presents with severe chest pain radiating to the back or between the shoulder blades. The chest x-rays in such cases often show a widened mediastinum.

Patients with cocaine intoxication often present with psychomotor agitation. In such patients, like this one, IV diazepam is given until the agitation is controlled.

Calcium-channel blockers (diltiazem and verapamil) are used as adjunctive therapy in patients who continue to have ischaemic symptoms despite using nitroglycerin. It is not contraindicated in patients with acute cocaine intoxication.

Sublingual nitroglycerin is one of the first drugs administered to patients presenting with ischaemic chest pain. Both nitrates and calcium channel blockers are beneficial for cocaine-induced coronary artery vasoconstriction.

Discuss (5) Improve

Next question >

Cocaine

Cocaine is an alkaloid derived from the coca plant. It is widely used as a recreational stimulant. The price of cocaine has fallen sharply in the past decade resulting in cocaine toxicity becoming a

much more frequent clinical problem. This increase has made cocaine a favourite topic of question writers.

Mechanism of action

- cocaine blocks the uptake of dopamine, noradrenaline and serotonin

Adverse effects

- cardiovascular
  - coronary artery spasm → myocardial ischaemia/infarction
  - both tachycardia and bradycardia may occur
  - hypertension
  - QRS widening and QT prolongation
  - aortic dissection
- neurological
  - seizures
  - mydriasis
  - hypertonia
  - hyperreflexia
- psychiatric effects
  - agitation
  - psychosis
  - hallucinations
- others
  - ischaemic colitis is recognised in patients following cocaine ingestion. This should be considered if patients complain of abdominal pain or rectal bleeding
  - hyperthermia
  - metabolic acidosis
  - rhabdomyolysis

Management of cocaine toxicity

- in general, benzodiazepines are generally first-line for most cocaine-related problems
- chest pain:
  - benzodiazepines + glyceryl trinitrate
  - if myocardial infarction develops then primary percutaneous coronary intervention
- hypertension: benzodiazepines + sodium nitroprusside
- the use of beta-blockers in cocaine-induced cardiovascular problems is a controversial issue
  - the American Heart Association issued a statement in 2008 warning against the use of beta-blockers (due to the risk of unopposed alpha-mediated coronary vasospasm) but many cardiologists since have questioned whether this is valid
  - if a reasonable alternative is given in an exam it is probably wise to choose it

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Cocaine Dependence

Osmosis - YouTube 4 2

Report broken media

Score: **15.1%**

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A 28-year-old woman was admitted to the Emergency Department drowsy and unwell after a suspected suicide attempt at home. The patient was under the care of the community psychiatry and had been receiving treatment for schizoaffective disorder. There was no other known past medical history. The paramedics bringing the patient to hospital also had brought the medications they found in the patient's home including quetiapine, levomepromazine, zopiclone and oxazepam. The paramedics reported that they had not seen evidence of empty medication packets or blister packs at the patient's home.

Initial assessment of the patient was unremarkable except for reduced consciousness level. Basic observations at presentation are listed below.

- Blood pressure: 150/79 mmHg
- Heart rate: 89 bpm
- Respiratory rate: 20 / min
- O2 saturations (15 L O2):100 %
- Glasgow coma score: M5 V3 E2
- Blood glucose: 7.0 mmol / L
- Temperature: 36.8ºC

Results from an arterial blood sample (15 L O2) were as follows.

pH	7.19
PaCO2	3.3 kPa
PaO2	21.2 kPa
Bicarbonate	8.9 mmol / L (reference 20.0-26.0)

Sodium	142 mmol / L
Potassium	3.6 mmol / L
Calcium	2.13 mmol / L (reference 2.20-2.60)
Chloride	110 mmol / L (reference 99-108)
Urea	5.2 mmol / L
Creatinine	110 micromol / L
Lactate	26 mmol / L
Plasma osmolality	380 mmol / Kg (reference 280-295)
Haemoglobin	12.0 g / dL

Based on the above blood results, what is the most likely cause of the patient's reduced consciousness level?

- Ethanol intoxication
- Benzodiazepine overdose
- Ethylene glycol intoxication
- Quetiapine overdose
- Chronic renal failure

Submit answer

Reference ranges

Score: **0%**

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Plasma osmolality	380 mmol / Kg (reference 280-295)
Haemoglobin	12.0 g / dL

Based on the above blood results, what is the most likely cause of the patient's reduced consciousness level?

Ethanol intoxication	9%
Benzodiazepine overdose	4%
Ethylene glycol intoxication	82%
Quetiapine overdose	5%
Chronic renal failure	0%

The patient is unwell with a severe lactic acidosis. However, there is no clinical evidence for conditions that may cause this state (such as shock, hypoxia, sepsis or lactate-inducing drugs). Calculation of the anion gap and osmolality gap provide further clues to the likely cause.

Anion gap = Sodium - (Chloride + Bicarbonate) = 23.1 mmol / L (reference 5-11)

Osmolality gap = Measured osmolality - ((2*Sodium) + Urea + glucose)) = 83.8 mmol / Kg (reference < 6)

A severe lactic acidosis may cause a small osmolality gap (< 10 mmol / Kg) but is not consistent with the high value in this case). Possible causes of a high osmolality gap include pseudohyponatraemia (in extreme hyperproteinaemia or hyperlipidaemia), chronic renal failure or excess of toxic alcohols or glycols.

However, in the case of ethanol intoxication or pseudohyponatraemia the osmolality gap is not associated with a metabolic acidosis. Given that the blood results do not suggest evidence of chronic renal failure, the most likely cause is ethylene glycol intoxication. Testing for serum levels of methanol and ethylene glycol would confirm the diagnosis. Appropriate treatment is with ethanol and haemodialysis.

There is no evidence from the collateral history that the patient had taken an overdose of her prescription medications and this would not explain the metabolic abnormalities.

Oostvogels R, Kemperman H, Hubeek I, ter Braak E. The importance of the osmolality gap in ethylene glycol intoxication. BMJ 2013;347:f6904.

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **15.1%**

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A 17-year-old girl is brought to the Emergency Department by ambulance after her drink was 'spiked' at a party. Her friends recall her complaining that she felt like she was having an 'out of body experience' and that everything she touched seemed artificial. Over the next hour, she became increasingly distressed, slurring her words and appearing off-balance when walking.

Her past medical history is unremarkable. Her friends state that she does not usually partake in recreational drug use and had only consumed 2 or 3 alcoholic drinks during the course of the evening.

Examination reveals a slim young female with a temperature of 37.3, a pulse of 113bpm and a blood pressure of 137/76mmHg. Her pupils measure 5mm in diameter and she is GCS 14/15 due to persistent confusion. Her chest is clear and her heart sounds are unremarkable. Her abdomen is soft and non-tender. Neurological examination reveals bi-directional nystagmus and truncal ataxia with no focal weakness. Her deep tendon reflexes are brisk and symmetrical.

Which drug is she most likely to have been exposed to?

- Ecstasy
- Methoxetamine
- Cocaine
- Gamma-hydroxybutyric acid (GHB)
- Nexus (2C-B)

Submit answer

Reference ranges

Score: **0%**

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Which drug is she most likely to have been exposed to?

Ecstasy	31%
Methoxetamine	16%
Cocaine	6%
Gamma-hydroxybutyric acid (GHB)	31%
Nexus (2C-B)	16%

Both ecstasy and cocaine could cause the tachycardia, hypertension, mydriasis and marginally elevated temperature described in the above scenario. The 'dead giveaway' however, is the presence of dissociative symptoms in combination with an acute cerebellar syndrome, pointing strongly towards methoxetamine intoxication.

Methoxetamine is an analogue of ketamine; a dissociative anaesthetic that acts as a NMDA receptor antagonist. Its effects are typical of the adverse effects of ketamine and include tachycardia, hypertension, mydriasis, confusion and agitation. Nystagmus, dysarthria, and ataxia can be severe and persist for several days.

GHB is a relatively short-acting CNS depressant that causes euphoria followed by coma. Patients can often be roused by external stimuli before returning to a comatose state. Rapid, spontaneous recovery can occur and patients have been known to extubate themselves.

Nexus (2C-B) is a synthetic phenylethylamine with psychoactive and stimulant effects. It is typically snorted or ingested with the former typically causing intense nasal pain. Its effects on the CNS include euphoria, synaesthesia, hallucinations and increased visual and tactile sensation, and probably occur due to antagonism of the 5HT2 receptor. Nexus (2C-B) is also an alpha-1 receptor agonist and its use can lead to ischaemic complications.

Discuss (4) | Improve

Next question >

Novel psychoactive substances

Novel psychoactive substances is the medical term for the many new substances which are chemically related to established recreational drugs such as MDMA and cannabis. They are often referred to as 'legal highs' although this is a misnomer in the UK, as their distribution and sale have been illegal since 2016.

The information below describes some of the common types:

Stimulants

- similar to MDMA, amphetamines and cocaine, resulting in increased levels of serotonin, dopamine and noradrenaline, resulting in a 'high' and feeling of euphoria
- a common example is a stimulant NPS is mephedrone ('bath salts','M-CAT'.'meow meow'). It is a cathinone and structurally similar to khat, a plant found in East Africa
- another example is benzylpiperazine ('Exodus', 'Legal X', 'Legal E')
- typically swallowed as a pill/powder ('bombing') or snorted
- adverse effect profile similar to MDMA/cocaine, with the risk of serotonin syndrome

Cannabinoids

- termed synthetic cannabinoid receptor agonists
- commonly referred to as 'spice'
- typically sprayed on to herbal mixtures which are then smoked. Also available in liquid form which is then inhaled using e-cigarettes
- similar adverse effects to cannabis

Hallucinogenics

- can be either dissociatives and psychedelics
- dissociatives produce a similar effect to ketamine, with a sense of not being connected to the physical body or time. A common dissociative NPS is methoxetamine ('mexxy')
- psychedelics have a similar effect to LSD although NPS versions may also be a stimulant

Depressant

- can be either opioid or benzodiazepine-based
- usually taken as a pill or a powder
- often structurally very similar to the original drug class, hence the adverse effects are similar
- benzodiazepine NPS often have a significantly longer half-life

Other substances include:

- Gamma-hydroxybutyric acid (GHB) and gamma-butyrolactone (GBL): 'G', 'Geebs' or 'Liquid Ecstasy'
- GHB causes respiratory depression. When taken with other respiratory depressants, most commonly alcohol, this can be potentially life threatening
- Nitrous oxide: 'Hippie crack'

For a more detailed overview please see the excellent review in BMJ 2017;356:i6848

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 35-year-old woman is referred to oncology. She has recently been diagnosed with metastatic breast cancer, which is HER2 positive. She has no other past medical history. She does not smoke or drink alcohol. She is an artist and lives with her husband and two young children.

She is commenced on trastuzumab.

The concurrent administration of which medication or medications should be avoided?

- Carboplatin and paclitaxel
- Docetaxel and cyclophosphamide
- Doxorubicin and cyclophosphamide
- Pertuzumab
- Pertuzumab and hyaluronidase-zzxf

Submit answer

Reference ranges

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She is commenced on trastuzumab.

The concurrent administration of which medication or medications should be avoided?

Carboplatin and paclitaxel	9%
Docetaxel and cyclophosphamide	12%
Doxorubicin and cyclophosphamide	66%
Pertuzumab	8%
Pertuzumab and hyaluronidase-zzxf	6%

The combination of trastuzumab + anthracyclines (both often used for breast cancer) is particularly cardiotoxic

Important for me Less important

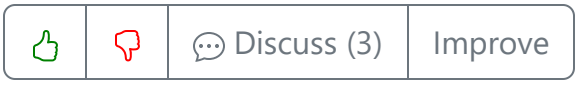
Doxorubicin and cyclophosphamide is the correct answer. This combination is particularly cardiotoxic and concurrent administration of trastuzumab and anthracyclines (doxorubicin) is not recommended.

Carboplatin and paclitaxel are not correct. This is a possible combination of chemotherapeutic agents that could be used in conjunction with trastuzumab to treated metastatic HER2 positive breast cancer.

Docetaxel and cyclophosphamide is not the right answer. This is a possible combination of chemotherapeutic agents that could be used in conjunction with trastuzumab to treated metastatic HER2 positive breast cancer.

Pertuzumab is incorrect. Research shows that adding pertuzumab to trastuzumab and chemotherapy as part of first-line therapy for HER2-positive metastatic breast cancer improves mortality.

Pertuzumab and hyaluronidase-zzxf is incorrect. This can be combined with trastuzumab to be administered as a single medication combination and is used in the treatment of metastatic HER2 positive breast cancer.



Next question

Trastuzumab

Trastuzumab (Herceptin) is a monoclonal antibody directed against the HER2/neu receptor. It is used mainly in metastatic breast cancer although some patients with early disease are now also given trastuzumab.

Adverse effects

- flu-like symptoms and diarrhoea are common
- cardiotoxicity
  - more common when anthracyclines have also been used
  - an echo is usually performed before starting treatment

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Biologics

9 4

Trastuzumab in the management of early and advanced stage breast cancer

Suggest link Report broken link

Score: **15.1%**

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A 19-year-old woman is referred to the acute medical team after presenting to the emergency department. The patient reports taking an overdose of paracetamol that evening, following an argument with her mother. She immediately regretted taking the overdose and asked her mother to take her to the hospital. She is now very upset and anxious that she has caused herself serious harm and is requesting treatment.

The patient took just under two packets of 500 mg paracetamol tablets (an estimated 28 tablets), 40 minutes prior to presentation to hospital. She denied any history of mental or physical health problems, and had never taken an overdose before. She took no regular medications and did not drink alcohol.

Examination revealed an alert but anxious young woman with observations within normal physiological parameters, and no abnormal physical signs.

Haemoglobin	128 g / dL
Mean cell volume	95.0 fl
White cell count	8.4 x 10>3 / microlitre
Platelets	198 x 10>3 / microlitre
Urea	4.3 mmol / L
Creatinine	67 micromol / L
Sodium	137 mmol / L
Potassium	4.3 mmol / L
Albumin	45 g / L (reference 35-50)
Alkaline phosphatase	56 U / L (reference 35-100)
ALT	34 U / L (reference 3-36)
Bilirubin	15 micromol / L (reference < 26)
International normalised ratio	1.1 (reference 0.8-1.2)

What is the most appropriate management of the patient's paracetamol overdose?

- Treatment with activated charcoal if 4-hour paracetamol level > 100 mg / L
- Immediate treatment with acetylcysteine

- Haemodialysis
- Immediate treatment with methionine
- Immediate treatment with activated charcoal

Submit answer

Reference ranges

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What is the most appropriate management of the patient's paracetamol overdose?

Treatment with activated charcoal if 4-hour paracetamol level > 100 mg / L 7%

Immediate treatment with acetylcysteine 23%

Haemodialysis	1%
Immediate treatment with methionine	1%
Immediate treatment with activated charcoal	68%

The patient has taken a large (> 10 g) paracetamol overdose and then presented to the hospital very promptly (< 2 hours). For such patients, activated charcoal should be considered as a first-line therapy, if the risk of aspiration is low. In practice, this means that the patient is either fully compliant with treatment (as in this example) or is intubated.

No randomised controlled trials have examined the role of activated charcoal in paracetamol overdose. However, a cohort study of patients who ingested > 10 g paracetamol, found that treatment with charcoal within two hours was associated with reduced need for acetylcysteine. If this link is assumed to be causal, then number needed to treat to avoid further treatment was 7. In addition, two randomised controlled trials have shown that activated charcoal is very safe.

The use of activated charcoal also increases elimination of anti-epileptic and contraceptive medications, and precautions should be taken against the short-term risk of treatment failure.

Treatment decisions regarding paracetamol poisoning antidote (acetylcysteine or methionine) should be guided by the 4-hour paracetamol level and reference to paracetamol poisoning nomogram. There is no benefit to treatment prior to the 4-hour paracetamol level result; hepatotoxicity following paracetamol toxicity does not occur until glutathione stores are depleted, around 8 hours after the overdose.

Haemodialysis removes only a small proportion of ingested paracetamol, as well as removing acetylcysteine. Therefore, it is not a therapeutic option in paracetamol overdose.

Buckley N, Dawson A, Isbister G. Treatments for paracetamol poisoning. BMJ 2016;353:i2579.

Discuss (6) Improve

Next question ›

Paracetamol overdose: management ★

The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk

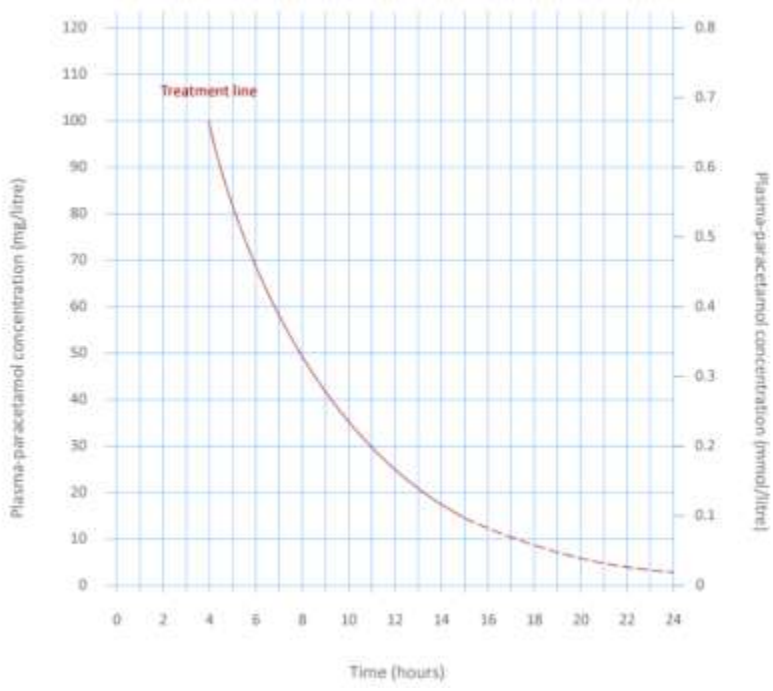
factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

Next question

Textbooks

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Links

Medicines and Healthcare products Regulatory Agency

3 3

2012 Paracetamol overdose update

Suggest link Report broken link

Score: **15.1%**

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A 78-year-old man with a history of atrial fibrillation, diabetes, hypertension and peripheral neuropathy was referred to the anticoagulation clinic by his General Practitioner due to difficulty controlling the international normalised ratio (INR). He is currently on 12 mg of warfarin and the dose had to be repeatedly increased as his INR has been in the subtherapeutic range in the last month.

Which one of the following medication may be responsible for the above?

- Omeprazole
- Carbamazepine
- Cimetidine
- Valproate
- Ciprofloxacin

Submit answer

Reference ranges

Score: **0%**

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Which one of the following medication may be responsible for the above?

Omeprazole	7%
Carbamazepine	67%
Cimetidine	5%
Valproate	10%
Ciprofloxacin	12%

Carbamazepine is a P450 enzyme inductor

Important for me Less important

Carbamazepine is a hepatic enzyme inducer and may reduce the effectiveness of warfarin, whereas the others are enzyme inhibitors.

Others to remember are:

P450 enzyme inducers	P450 enzyme inhibitors
Phenytoin	Amiodarone
Rifampicin	Ketoconazole
Alcohol	Erythromycin
	Isoniazid
	Sulphonamides

P450 enzyme inducers	P450 enzyme inhibitors
	Allopurinol

Discuss (3) Improve

Next question

P450 enzyme system

Induction usually requires prolonged exposure to the inducing drug, as opposed to P450 inhibitors, where effects are often seen rapidly

Inducers of the P450 system include

- antiepileptics: phenytoin, carbamazepine
- barbiturates: phenobarbitone
- rifampicin
- St John's Wort
- chronic alcohol intake
- griseofulvin
- smoking (affects CYP1A2, reason why smokers require more aminophylline)

Inhibitors of the P450 system include

- antibiotics: ciprofloxacin, erythromycin
- isoniazid
- cimetidine,omeprazole
- amiodarone
- allopurinol
- imidazoles: ketoconazole, fluconazole
- SSRIs: fluoxetine, sertraline
- ritonavir
- sodium valproate
- acute alcohol intake
- quinupristin

AI + Q 123

Next question

Textbooks

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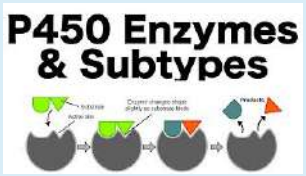
Media



P450 Enzyme System (Inducers, Inhibitors, & Subtypes)

Dirty Medicine - YouTube 1 0

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A 62-year-old man presents to the emergency department after drinking a small amount of methanol in an attempt to end his life. His observations are as follows: heart rate 110 beats per minute, blood pressure 130/85 mmHg, respiratory rate 22 breaths per minute, temperature 37.5ºC, and SpO2 95% on air. A fundoscopic examination is normal.

Blood results are as follows:

Hb	120 g/L	Male: (135-180) Female: (115 - 160)
Platelets	165 * 10^9/L	(150 - 400)
WBC	14.8 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	2.8 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Glucose	4.2 mmol/L	(3.9 - 5.6)
Serum osmolarity	305 mOsm/kg	(275 - 295)

You start the patient on fomepizole.

What additional treatment is required?

- Activated charcoal
- Ethanol
- Folic acid
- Folinic acid
- Haemodialysis

Submit answer

Reference ranges

Score: **0%**

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You start the patient on fomepizole.

What additional treatment is required?

- Activated charcoal 4%
- Ethanol 26%
- Folic acid 8%
- **Folinic acid 53%**
- Haemodialysis 9%

Folinic acid can be used in methanol poisoning to reduce ophthalmological complications

The patient has clinical and biochemical features of methanol toxicity.

The osmolar gap = osmolality (measured) - osmolarity (calculated). A normal osmolar gap should be < 10.

The calculated osmolarity in this case = (2 x [Na+]) + [glucose] + [urea]) = 277 mOsm/kg.

Therefore the osmolar gap = 305 - 277 = 28 mOsm/kg.

The patient thus has a high osmolar gap which indicates the presence of an abnormal solute present in significant amounts.

Causes of a high osmolar gap include:

- Mannitol
- Methanol
- Ethylene glycol

Folinic acid is correct. The clinical and biochemical features support a diagnosis of methanol poisoning. For patients with methanol intoxication, early administration of fomepizole will minimise the further metabolism of methanol and the development of clinical and metabolic complications. The addition of folinic acid can help reduce the accumulation of toxic byproducts in the eyes thereby reducing the associated ophthalmological complications.

Activated charcoal is incorrect. Activated charcoal is of no use since it does not adsorb significant quantities of methanol.

Ethanol is incorrect. Ethanol results in competitive inhibition of methanol thereby slowing down formic acid formation. Fomepizole is the preferred antidote since, unlike ethanol, it does not require regular monitoring of blood concentrations or cause inebriation.

Folic acid is incorrect. It is folinic acid rather than folic acid which has the best evidence base to reduce the ophthalmological complications of methanol.

Haemodialysis is incorrect. Haemodialysis has a role in severe or refractory cases. Indicators of severe disease include a methanol concentration greater than 500 mg/L, visual disturbance, features of CNS toxicity, severe metabolic acidosis and renal failure.

Discuss (1) Improve

Next question

Methanol poisoning

Methanol poisoning causes both the effects associated with alcohol (intoxication, nausea etc) and also specific visual problems, including blindness. These effects are thought to be secondary to the accumulation of formic acid. The actual pathophysiology of methanol-associated visual loss is not fully understood but it is thought to be caused by a form of optic neuropathy

Management

- fomepizole (competitive inhibitor of alcohol dehydrogenase) or ethanol
- haemodialysis
- cofactor therapy with folinic acid to reduce ophthalmological complications

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane 1 5

Toxic Alcohol Ingestion

Suggest link Report broken link

Score: **15.1%**

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A 57-year-old man attends neurology clinic. He has recently been diagnosed with Parkinson's disease and has agreed to start treatment with cabergoline as monotherapy. He has had a full examination, lung function tests, routine blood tests, chest X-ray and an echocardiogram.

Apart from a clinical review, what investigation is most important to arrange regularly to monitor for complications?

- Chest X-ray
- FBC
- U&E
- Echocardiogram
- LFT

Submit answer

Reference ranges

Score: **0%**

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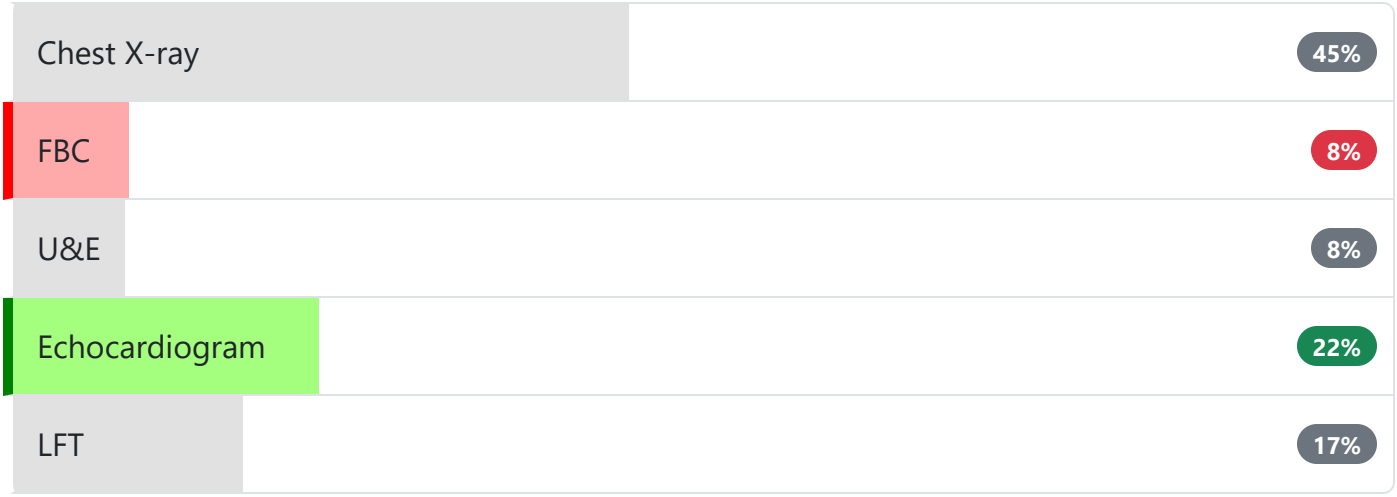

The correct answer is echocardiogram. Cabergoline is a dopaminergic drug which can be used to treat Parkinson's disease. Dopaminergic drugs are associated with fibrotic reactions including pulmonary, retroperitoneal and pericardial fibrosis, but cabergoline also assocaited with development of new valvulopathy. The BNF recommends establishing a baseline set of investigations and then to monitor clinically for dyspnoea, persistent cough, chest pain, cardiac failure and abdominal pain or tenderness.

Source:

Committee, Joint Formulary. British National Formulary (BNF) No. 72. London: Pharmaceutical, 2016.

Dopamine receptor agonists

Indications

- Parkinson's disease
- prolactinoma/galactorrhoea
- cyclical breast disease
- acromegaly

Currently accepted practice in the management of patients with Parkinson's disease is to delay treatment until the onset of disabling symptoms and then to introduce a dopamine receptor agonist. If the patient is elderly, L-dopa is sometimes used as an initial treatment

Overview

- e.g. bromocriptine, ropinirole, cabergoline, apomorphine
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline, pergolide*) have been associated with pulmonary, retroperitoneal and cardiac fibrosis. The Committee on Safety of Medicines advice that an ESR, creatinine and chest x-ray should be obtained prior to treatment and patients should be closely monitored

Adverse effects

- nausea/vomiting
- postural hypotension
- hallucinations
- daytime somnolence

*pergolide was withdrawn from the US market in March 2007 due to concern regarding increased incidence of valvular dysfunction

AI + Q 123

Next question >

B I A T

Textbooks

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Score: **15.1%**

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A 52-year-old man is brought into the emergency department resuscitation room. He was found collapsed by his wife 40 minutes ago. He was last seen 3 hours prior. His wife is present, who informs you his past medical history consists of depression, osteoarthritis, sciatica and ischaemic heart disease.

His Glasgow coma scale was 13/15. Nystagmus was noted.

Observations: respiratory rate 28 breath/minute, oxygen saturations 100% on 15 litres of oxygen, heart rate 112 beats/minute, blood pressure 142/92mmHg. Electrocardiogram (ECG) is sinus tachycardia, but otherwise normal.

An arterial blood gas is done and bloods are sent to the lab.

Hb	143 g/L	Male: (135-180) Female: (115 - 160)
Platelets	383 * 10^9/L	(150 - 400)
WBC	13.2 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	5 mmol/L	(3.5 - 5.0)
Urea	9 mmol/L	(2.0 - 7.0)
Creatinine	135 µmol/L	(55 - 120)
CRP	14 mg/L	(< 5)
Glucose	6 mmol/L	(4-7)
Osmolality	322mOsm/kg	(275 - 295)
pH	7.31	(7.35-7.45)
pO2	26 kPa	(10-14)
pCO2	2.9 kPa	(4.5-6)
$HCO3^-$	12 mmol/L	(22-28)
Cl^-	95 mmol/L	(95-105)

What is the most likely cause of this man's presentation?

- Amitriptyline

- [ ] Citalopram
- [ ] Codeine
- [ ] Ethylene glycol
- [ ] Mirtazapine

Submit answer

Reference ranges

Score: **0%**

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What is the most likely cause of this man's presentation?

Amitriptyline 7%

Citalopram	3%
Codeine	1%
Ethylene glycol	88%
Mirtazapine	1%

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

This question involves toxicology and utilising the anion gap and osmolar gap helps reach the diagnosis. The presentation of reduced GCS is non-specific and can occur in many toxidromes. From his observations, we can see that he has a high respiratory rate, despite normal saturations.

Assessing his arterial gas, he has a partially compensated metabolic acidosis. His high respiratory rate is helping to blow off CO2 to compensate. The anion gap can be calculated as 33 or 38 (depending on whether you include potassium in your calculation). This is high.

When osmolality is stated in a question regarding toxicology it is often worth calculating the osmolar gap - the difference between measured and calculated osmolality. Calculated osmolality is $2Na^+$ + glucose + urea. The normal range of the osmolar gap is less than 10. In this case, it's 27.

Common causes of a high osmolar gap in toxicology are ethylene glycol, ethanol and methanol. Out of these three, only ethylene glycol is an option and so this is the most likely answer.

Paracetamol and selective serotonin reuptake inhibitors may cause high anion gap metabolic acidosis indirectly by causing acute kidney injury, but are not typically associated with a high osmolar gap.

Amitriptyline is a tricyclic. Within 1-2 hours of onset of toxicity they tend to cause hypotension and tachycardia, classically broad-complex. This progresses to seizures and reduced level of consciousness. This patient is not hypotensive and the ECG is not broad-complex so this is less likely.

Codeine is an opioid and so you expect to find respiratory depression if this was the case in this patient.

Discuss Improve

Next question

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

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Score: **15.1%**

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You are in the general medical clinic. A 37-year-old male has been referred by his GP with dyspnoea. His symptoms started about 5 months ago with dyspnea mostly on exertion which is progressively getting worse. He also has a dry cough for last 2 months. There is no orthopnoea or paroxysmal nocturnal dyspnoea. There is also no history of chest pain. His past history includes Hodgkin's lymphoma which was successfully treated 2 years ago with ABVD regimen. He is currently not taking any medications. He works in a printing press and does not smoke. On examination, there is no raised JVP or ankle oedema. Auscultation revealed normal heart sounds and bilateral fine crackles. Results of his investigations are as follows:

Hb	125 g/l	Na^+	139 mmol/l	Bilirubin	12 µmol/l
Platelets	188 * 10^9/l	K^+	4.1 mmol/l	ALP	95 u/l
WBC	6.7 * 10^9/l	Urea	4.3 mmol/l	ALT	15 u/l
Neuts	3.5 * 10^9/l	Creatinine	86 µmol/l	γGT	45 u/l
Lymphs	2.2 * 10^9/l	ESR	25 mm/hr	Albumin	34 g/l
Eosin	0.3 * 10^9/l				

Chest x-ray normal

What is the next best investigation for evaluation of this patient?

- Bronchoalveolar lavage (BAL)
- Mantoux test
- Echocardiogram
- Chest ultrasound
- Pulmonary function tests

Submit answer

Reference ranges

Score: **0%**

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Back to top

You are in the general medical clinic. A 37-year-old male has been referred by his GP with dyspnoea. His symptoms started about 5 months ago with dyspnea mostly on exertion which is progressively getting worse. He also has a dry cough for last 2 months. There is no orthopnoea or paroxysmal nocturnal dyspnoea. There is also no history of chest pain. His past history includes Hodgkin's lymphoma which was successfully treated 2 years ago with ABVD regimen. He is currently not taking any medications. He works in a printing press and does not smoke. On examination, there is no raised JVP or ankle oedema. Auscultation revealed normal heart sounds and bilateral fine crackles. Results of his investigations are as follows:

Hb	125 g/l	Na^+	139 mmol/l	Bilirubin	12 µmol/l
Platelets	188 * 10^9/l	K^+	4.1 mmol/l	ALP	95 u/l
WBC	6.7 * 10^9/l	Urea	4.3 mmol/l	ALT	15 u/l
Neuts	3.5 * 10^9/l	Creatinine	86 µmol/l	γGT	45 u/l
Lymphs	2.2 * 10^9/l	ESR	25 mm/hr	Albumin	34 g/l
Eosin	0.3 * 10^9/l				

Chest x-ray normal

What is the next best investigation for evaluation of this patient?

Bronchoalveolar lavage (BAL)	9%
Mantoux test	4%
Echocardiogram	23%
Chest ultrasound	1%
Pulmonary function tests	**63%**

Bleomycin may cause pulmonary fibrosis

Important for me Less important

History is suggestive of interstitial lung disease most likely secondary to bleomycin and pulmonary function tests will aid in diagnosis by showing a restrictive pattern. Bleomycin is part of ABVD

regimen and known to cause pulmonary fibrosis. That's why baseline pulmonary function testing is important in patients receiving bleomycin.

BAL is an invasive procedure with non-specific findings in cases of drug-induced interstitial lung disease. Non-invasive investigations like pulmonary function tests and high-resolution CT (HRCT) are carried out first before proceeding for invasive tests.

Mantoux test can help exclude tuberculosis but tuberculosis is not the most likely diagnosis in the given scenario.

An echocardiogram can be used to exclude dyspnoea due to heart failure, however, there is no history of orthopnoea or paroxysmal nocturnal dyspnoea, and no findings of raised JVP, abnormal heart sounds or ankle oedema.

Chest ultrasound is not helpful unless a pleural effusion was suspected.

Discuss (11) | Improve

Next question ›

Drugs causing lung fibrosis

Causes

- amiodarone
- cytotoxic agents: busulphan, bleomycin
- anti-rheumatoid drugs: methotrexate, sulfasalazine
- nitrofurantoin
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline, pergolide)

123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 48-year-old farmer is found collapsed in his workshop having left a suicide note and consumed unknown weedkillers and insecticides from his store. On arrival in the Emergency department, his Glasgow coma scale is 10, pupils are pinpoint and poorly reactive. His blood pressure is 100/70 mmHg, his pulse is 88 beats per minute and regular. He moans incomprehensibly when you palpate his abdomen. You are concerned about the possibility of a paraquat overdose. How can you screen for it?

- Serum NADPH testing
- Serum free fatty acid testing
- Urine copper sulphate testing
- Urine ketone testing
- Urine dithionate testing

Submit answer

Reference ranges

Score: **0%**

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Serum NADPH testing	32%
Serum free fatty acid testing	7%
Urine copper sulphate testing	4%
Urine ketone testing	3%
Urine dithionate testing	55%

Qualitative confirmation of paraquat in the urine is easily performed in any laboratory. The main role of this test is to confirm or exclude exposure to paraquat. The presence of paraquat is confirmed by a colour change noted after the addition of a dithionite solution. The test is usually positive within six hours of a large ingestion and remains positive for several days. It's important not to wait for the results of the test before administering oral Fuller's earth.

The other options have no value in the evaluation of paraquat poisoning. Serum free fatty acids may rise where there is mitochondrial dysfunction, as may urinary ketones. Serum lactate is used in evaluation of metabolic dysfunction associated with cyanide poisoning.

Discuss (5) | Improve

Next question

Overdose and poisoning: management

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization with IV bicarbonate • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil The majority of overdoses are managed with supportive care only due to the risk of seizures with flumazenil. It is generally only used with severe or iatrogenic overdoses.
Tricyclic antidepressants	Management • IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity • arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics
Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine

Toxin	Treatment
	• in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit
Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF

Emergency treatment of poisoning

2 1

Royal College of Physicians

2012 Poisoning and self-harm update

1 1

Royal College of Physicians

2007 Acute poisoning on the medical admissions unit

1 2

Suggest link

Report broken link

Score: **15.1%**

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A 35-year-old man was assessed in endocrinology clinic after being referred by his GP for advice on management of the consequences of the patient's use of anabolic steroids. The patient reported that he had been using anabolic steroids intermittently for the previous 10 years to augment his weight-training regime. Having recently become aware of the potential adverse health consequences , the patient now wished to know how he could safely stop his anabolic steroid use.

The patient described his pattern of anabolic steroid use: typically, he had been taking a 'cycle' of one or more oral synthetic testosterone derivatives for between 6-12 weeks, prior to a 3-4 week break in his use of steroids. The patient stated this strategy was an attempt to minimise his risk of side effects. The patient had not received any medical supervision of his anabolic steroid use, relying instead on discussions with friends at his gym and information from online forums. The patient declined to disclose the source from which he had obtained his supply of medications.

The patient stated that he believed his anabolic steroid use had contributed to his male pattern baldness and also had caused intermittent breakouts of acne on his chest and face. The patient was not aware of any other symptoms related to his steroid use, although stated that he was concerned about possible lasting cardiac side effects. The patient had no other significant medical history and took no other regular medications. He did not consume cigarettes, alcohol or recreational drugs.

General examination of the patient revealed a muscular and lean adult male in apparent good health. A cardiovascular system examination noted a forceful but not displaced apex beat. Mild gynaecomastia was present but gastrointestinal examination was otherwise unremarkable. Using an orchidometer the patient's testicular volume was estimated as 16 ml. Although anxious about the possible health consequences of his anabolic steroid use, the patient did not seem to be significantly depressed or anxious.

Haemoglobin	173 g / dL
Sodium	148 mmol / L
Potassium	3.6 mmol / L
Alkaline phosphatase	135 U / L (reference 35-100)
ALT	32 U / L (reference 3-36)
Bilirubin	20 micromol / L (reference < 26)
Prolactin	469 mU / L (reference 80 - 400)
Luteinising hormone	0.9 IU / L (reference 1.8 - 8.6)

Follicle-stimulating hormone	1.2 mU / ml (reference 1.5 - 12.4)
Fasting LDL cholesterol	4.1 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.0 mmol / L (reference > 1.2)
HbA1C	45 mmol / mol (reference < 42)

What is the appropriate advice to the patient regarding the safe cessation of anabolic steroid use?

- Convert patient to prescribed testosterone replacement and taper over 1 year
- Taper anabolic steroid use over period of 6 months
- Stop immediately, tapered withdrawal not required
- Taper anabolic steroid use over period of 6 weeks
- Convert patient to prescribed testosterone replacement and continue lifelong

Submit answer

Reference ranges

Score: **0%**

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HbA1C	45 mmol / mol (reference < 42)

What is the appropriate advice to the patient regarding the safe cessation of anabolic steroid use?

- Convert patient to prescribed testosterone replacement and taper over 1 year — 20%
- Taper anabolic steroid use over period of 6 months — 15%
- Stop immediately, tapered withdrawal not required — 35%
- Taper anabolic steroid use over period of 6 weeks — 27%
- Convert patient to prescribed testosterone replacement and continue lifelong — 3%

The patient has typical side effects and examination findings associated with long-standing anabolic steroid use. These include male pattern baldness, acne, gynaecomastia and testicular atrophy (typical adult male testicular volume around 25 mL). His blood tests also demonstrate typical abnormalities including erythropoiesis, hypernatraemia, hypokalaemia, cholestatic liver function tests, deranged lipid profile, evidence of elevated serum glucose and typical hormone profile abnormalities.

Discuss (4) | Improve

Next question >

Anabolic steroid use

Anabolic steroid use is associated with several serious long-term health consequences. Cardiac morbidity and mortality are increased by anabolic steroid use, although the precise mechanism of this effect is unclear. Hepatic side effects also occur secondary to chronic vascular injury: these include hepatocellular carcinoma and hepatic adenoma. Psychiatric illness is also commonly co-morbid with anabolic steroid use. Additionally, users who inject anabolic steroids have an increased risk of blood-borne viruses if needles are shared between individuals.

Due to the above concerns, patients should be strongly counselled to stop using anabolic steroids. There is no requirement for tapering of doses. Many of the above blood test abnormalities normalise once anabolic steroid consumption ceases. The expert recommendation is for lifelong monitoring for potential complications, initially annually with frequency reducing once blood markers normalise and in the absence of apparent adverse effects.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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Back to top

A 50 year-old man presents to the medical assessment unit with a two day history of nausea and vomiting. His notes are currently not available to you, and the patient is unable to recall his medical problems. He is currently an inpatient at the local psychiatry facility.

On examination, he appears drowsy with an ataxic gait. Neurological examination reveals normal tone with generalised reduced power. He also has a coarse tremor when his hands are outstretched.
Cardiorespiratory examination reveals is unremarkable.

ECG- sinus arrhythmia at 90 beats per minute. T wave inversion in leads V1-V3.

Observations

Blood pressure- 150/90 mmHg
Heart rate- 90 beats per minute
Respiratory rate- 16 breaths per minute
Oxygen saturations- 99% on room air
Temperature- 37.3ºC

What is the most likely cause of his symptoms?

- Lithium toxicity
- Alcohol withdrawal
- Serotonin syndrome
- Neuroleptic malignant syndrome
- Tricyclic antidepressant overdose

Submit answer

Reference ranges

Score: **0%**

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Blood pressure- 150/90 mmHg
Heart rate- 90 beats per minute
Respiratory rate- 16 breaths per minute
Oxygen saturations- 99% on room air
Temperature- 37.3ºC

What is the most likely cause of his symptoms?

Lithium toxicity	77%
Alcohol withdrawal	7%
Serotonin syndrome	5%
Neuroleptic malignant syndrome	4%
Tricyclic antidepressant overdose	6%

Lithium salts are used in the prophylaxis and treatment of bipolar disorder, refractory depression and aggressive or self-harming behaviour.

Lithium has a narrow therapeutic range (0.5-1 mmol/l). The likelihood of toxicity increases with increasing serum lithium levels. Toxic effects are seen with a concentration of >2 mmol/l.

Very early features may be non-specific, such as apathy and restlessness

Early signs of toxicity (1-2 mmol/l)

- anorexia and vomiting
- ataxia and dysarthria
- nystagmus
- blurred vision
- coarse tremor
- drowsiness

Late signs of toxicity (>2mmol/l)

- coma
- seizures
- acute renal failure
- death

Serotonin syndrome is a drug reaction to serotonergic agents, and ranges from mild to severe. It is characterised by a triad of autonomic hyperactivity (hyperthermia, tachycardia), neuromuscular abnormality (tremor) and mental state changes (confusion, coma).

Neuroleptic malignant syndrome is a rare reaction to neuroleptic drugs, often following initiation or increase in dose. It is characterised by recent neuroleptic use, hyperthermia, rigidity and autonomic dysfunction.

Discuss (5) | Improve

Next question >

Lithium toxicity ★

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure

- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- hyperreflexia
- acute confusion
- polyuria
- seizure
- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this
  - by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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Back to top

A 28-year-old man is brought into the emergency department by paramedics. He was found collapsed by his wife just outside the house. With the paramedics he was rousable and they initially suspected he was drunk.

On transfer, he has become more drowsy but is still talking on physical stimuli. Initial venous blood gas shows a metabolic acidosis with a high anion gap. Lab blood results show:

Hb	148 g/L	Male: (135-180) Female: (115 - 160)
Platelets	345 * 10^9/L	(150 - 400)
WBC	12.5 * 10^9/L	(4.0 - 11.0)
Na^+	135 mmol/L	(135 - 145)
K^+	5.2 mmol/L	(3.5 - 5.0)
Bicarbonate	18 mmol/L	(22 - 29)
Urea	12 mmol/L	(2.0 - 7.0)
Creatinine	148 µmol/L	(55 - 120)
Glucose	6.2 mmol/L	(4-10)
Osmolality	307 mOsm/kg	(275-295)
Paracetamol levels	<5 mg/L	(<5)
Salicylate levels	<5 mg/dL	(<5)

What is the most appropriate treatment?

- Chlorpromazine
- Cyproheptadine
- Ethanol
- Fomepizole
- Haemodialysis

Submit answer

Reference ranges

Score: **0%**

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Bicarbonate	18 mmol/L	(22 - 29)
Urea	12 mmol/L	(2.0 - 7.0)
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Osmolality	307 mOsm/kg	(275-295)
Paracetamol levels	<5 mg/L	(<5)
Salicylate levels	<5 mg/dL	(<5)

What is the most appropriate treatment?

Chlorpromazine	1%
Cyproheptadine	2%
Ethanol	9%
Fomepizole	87%
Haemodialysis	2%

Ethylene glycol toxicity management - fomepizole. Also ethanol / haemodialysis

Important for me Less important

Reduced consciousness is a non-specific symptom of poisoning. Looking at his blood tests show kidney injury, a mild inflammatory response and a high osmolality. The blood tests allow us to calculate whether there is an osmolar gap.

Calculated osmolarity = 2Na + glucose + urea
Calculated osmolarity = 2(135) + 6.2 + 12 = 288.2
Lab osmolality = 307

Osmolar gap = Osmolality (measured) - Osmolarity (calculated)
Osmolar gap = 307 - 288.2 = 18.8 (normal is <10)

There are several causes of an osmolar gap, including ethanol, isopropyl alcohol, methanol, ethylene glycol, and mannitol. **Fomepizole** is the treatment option used in the management of methanol or ethylene glycol poisoning which is likely to be the cause in this patient.

Chlorpromazine may be used in the management of serotonin syndrome. Although serotonin syndrome can cause metabolic acidosis, it would not explain the osmolar gap.

Cyproheptadine may be used in the management of serotonin syndrome. Although serotonin syndrome can cause metabolic acidosis, it would not explain the osmolar gap.

Ethanol is incorrect, as although it can be used in methanol and ethylene glycol poisoning, fomepizole is the superior antidote so is a better treatment if available. Ethanol has many issues as a treatment, including being difficult to dose and maintain appropriate levels.

Haemodialysis has a role in refractory cases of ethylene glycol toxicity. However, in this case, 1st-line management should be tried first, and therefore this answer is incorrect.

Discuss Improve

Next question >

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **15.1%**

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A 75-year-old man underwent a right hemicolectomy as radical treatment for a caecal adenocarcinoma. During the immediate post-operative period the patient was noted to be significantly confused and investigations to assess for a septic source were requested. Blood tests indicated new neutrophilia and thrombocytopenia. Noting that the patient had been started on enoxaparin for venous thrombosis prophylaxis on the first day after the operation, heparin induced thrombocytopenia antibodies were requested by the surgical junior team.

A specialist medical review was requested on day 6 post-operatively to assess the patient in the light of the results of these investigations. The patient was found to be back to his cognitive baseline and was starting to mobilise on the ward with the help of physiotherapy. Clinical examination was unremarkable except for the healing abdominal surgical incision.

Aside from his recent cancer diagnosis, the patient's past-medical history included only hypercholesterolaemia treated with simvastatin. He had no known allergies or drug sensitivities.

Blood result	Pre-operatively	Day 1 post-op	Day 4 post-op	Day 6 post-op
Platelets (x 10>3 / microlitre)	175	95	125	141
Neutrophils (x 10>3 / microlitre)	5.3	14.8	12.8	8.8
C-reactive protein (mg / L)	6	142	94	25
International normalised ratio	1.0	1.2	1.1	1.1

Chest x-ray: clear lung fields aside from minor bibasal atelectasis

Urine culture: no growth

CT abdomen: minimal abdominal free fluid, no evidence of focal collection, large bowel anastomoses intact

Heparin-induced thrombocytopenia antibodies: positive (moderate titre)

What is the likely cause for the patient's thrombocytopenia?

- Heparin induced thrombocytopenia
- Post-operative sepsis

- Post-operative thrombocytopenia
- Thrombotic thrombocytopenic purpura
- Disseminated intravascular coagulation

Submit answer

Reference ranges

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Heparin-induced thrombocytopenia antibodies: positive (moderate titre)

What is the likely cause for the patient's thrombocytopenia?

Heparin induced thrombocytopenia	35%
Post-operative sepsis	16%

Post-operative thrombocytopenia	43%
Thrombotic thrombocytopenic purpura	4%
Disseminated intravascular coagulation	2%

Heparin induced thrombocytopenia (HIT) antibodies have a high false positive rate and their interpretation needs to take into account careful consideration of the entire clinical picture. Decision tools to assess the likelihood of HIT such as the Warkentin probability scale are useful in this setting. This provides a pretest probability score to assist with interpretation of HIT antibody results based on the extent of the thrombocytopenia, the timing of onset relative to heparin administration, thrombotic events and the presence or absence of other causes of thrombocytopenia.

In this case, there are few clinical features consistent with HIT and a low pretest probability score would be calculated. The combination of this and moderate titre positive HIT antibodies is considered to exclude a HIT diagnosis.
In this patients case a post-operative thrombocytopenia is the most likely cause in the absence of evidence for other possible answers. The patients elevated inflammatory markers are secondary to surgery with no septic source identified on investigation. TTP can cause thrombocytopenia associated with delirium, although the patients clinical and haematological recovery make this an unlikely cause. DIC is excluded by the normal prothrombin time and improving clinical picture.

Linkins L-A. Heparin induced thrombocytopenia. BMJ 2014;349:7566.

Discuss (10) | Improve

Next question >

Heparin

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures

- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
    - direct thrombin inhibitor e.g. argatroban
    - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **15.1%**

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A 49-year-old woman has attended the clinic with a 3-month history of heat intolerance, weight loss, and intermittent diarrhoea.

She has a past medical history of paroxysmal ventricular tachycardia and type 2 diabetes mellitus. Her regular medications include amiodarone, metformin, and linagliptin.

On clinical examination, she is mildly tremulous with prominent skin capillaries. She is tachycardia but has no audible murmurs. A neck examination does not reveal a goitre.

Thyroid function tests are requested.

Free T4	42.1	11.0 - 23.0
TSH	0.05	0.35 - 5.00

Given the likely diagnosis, what is the best management option?

- Carbimazole
- Levothyroxine
- Potassium perchlorate
- Prednisolone
- Propylthiouracil

Submit answer

Reference ranges

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Thyroid function tests are requested.

Free T4	42.1	11.0 - 23.0
TSH	0.05	0.35 - 5.00

Given the likely diagnosis, what is the best management option?

Carbimazole	18%
Levothyroxine	1%
Potassium perchlorate	3%
Prednisolone	**74%**
Propylthiouracil	5%

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

Important for me Less important

The above case describes a patient with signs and symptoms commonly associated with hyperthyroidism. In combination with ongoing treatment with amiodarone, the diagnosis of amiodarone-induced thyrotoxicosis (AIT) can be made. AIT can be sub-divided into type 1 and type 2. This should not be confused with amiodarone-induced hypothyroidism which is thought to be due to the high iodine content of amiodarone.

Type 1 AIT is caused by excessive iodine from amiodarone causing increased thyroid hormone synthesis, whereas AIT type 2 is related to destructive thyroiditis. The two variations can be clinically distinguished by the presence (AIT type 1) or absence (AIT type 2) of a goitre. Given that the patient in the above scenario has no clinical findings of a goitre, AIT type 2 can be diagnosed. This is treated with corticosteroids, such as **prednisolone**.

Carbimazole is a well-recognised anti-thyroid medications commonly used in hyperthyroidism. It acts by reducing T3 and T4 production via thyroid peroxidase. It is an option in the management of AIT type 1.

Levothyroxine is used for thyroid hormone replacement in hypothyroidism and can be used in amiodarone-induced hypothyroidism.

Potassium perchlorate is an oxidising agent used as an anti-thyroid agent in thyrotoxicosis. It acts by competitively inhibiting iodine uptake in follicular thyroid cells. Along with carbimazole, it is a management option in type 1 AIT.

Although a treatment option in hyperthyroidism, **propylthiouracil** is not used in the management of amiodarone-induced thyrotoxicosis.

Discuss (1) | Improve

Next question >

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	AIT type 2
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

Score: **15.1%**

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A 35-year-old woman presents to the emergency department with agitation and sweating. She was recently started on paroxetine for depression. She takes St John's Wort over the counter. There is no history of substance abuse.

On examination, various muscle groups have sudden, brief involuntary twitching. She is diaphoretic, agitated and confused. There is hyperreflexia. She has a heart rate of 124 beats per minute and a temperature of 40ºC.

An ECG demonstrates sinus tachycardia.

Given the likely diagnosis, what is the appropriate treatment?

- Bisoprolol
- Co-amoxiclav
- Cyproheptadine
- Dantrolene
- Procyclidine

Submit answer

Reference ranges

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An ECG demonstrates sinus tachycardia.

Given the likely diagnosis, what is the appropriate treatment?

Bisoprolol	0%
Co-amoxiclav	0%
Cyproheptadine	69%
Dantrolene	23%
Procyclidine	7%

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

Important for me Less important

Cyproheptadine is correct. The patient has serotonin syndrome as a consequence of the interaction between St. John's Wort and paroxetine. Her presentation is clinically severe and she should be treated with cyproheptadine, which is a 5-HT2 receptor antagonist. Milder cases can be treated with benzodiazepines alone.

Bisoprolol is incorrect. Sinus tachycardia does not tend to need treatment with cardiovascular medications and the treatment should be against the underlying cause.

Co-amoxiclav is incorrect. While the patient has a fever, the clinical history of the recent combination of an SSRI and St. John's Wort along with myoclonus and hyperreflexia suggest serotonin syndrome rather than infection.

Dantrolene is incorrect. This is the treatment for malignant hyperthermia. This is also characterized by high fever and muscle spasms but occurs post exposure to inhalational anaesthetics or depolarising muscle relaxants.

Procyclidine is incorrect. This is an anti-cholinergic drug used in the treatment of drug-induced Parkinsonism.

Discuss (1) | Improve

Next question >

Serotonin syndrome ★

Causes

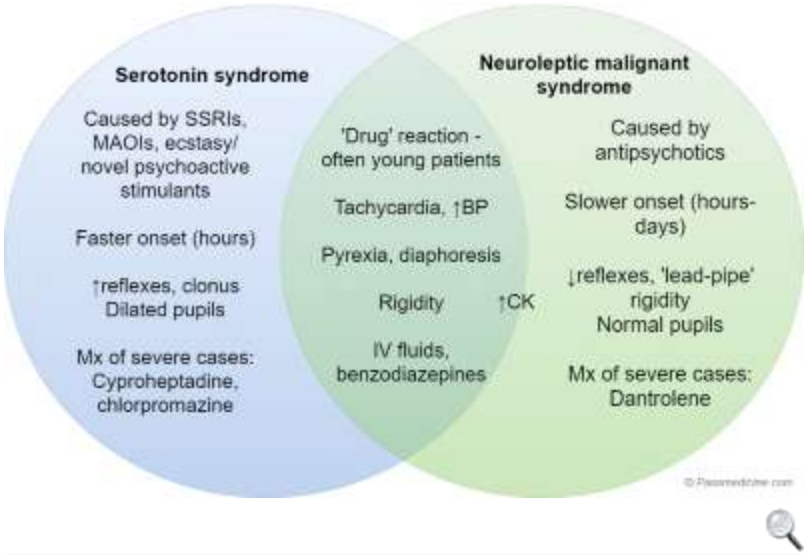
- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

Features

- neuromuscular excitation
  - hyperreflexia
  - myoclonus
  - rigidity
- autonomic nervous system excitation
  - hyperthermia
  - sweating
- altered mental state
  - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe 1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

Report broken media

Score: **15.1%**

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A 47-year-old man has been referred to the endocrinology clinic. He has a history of hypertension and bipolar disorder. He can recall his antihypertensives, lisinopril, and amlodipine, but cannot remember which medication he is on for his bipolar disorder.

Recently he has developed polydipsia and polyuria. His GP performed some blood tests and found elevated blood glucose. His HbA1c is sown below.

HbA1C	62 mmol/mol	42 - 48

What medication is the most likely cause?

- Aripiprazole
- Lithium
- Olanzapine
- Risperidone
- Sertraline

Submit answer

Reference ranges

Score: **0%**

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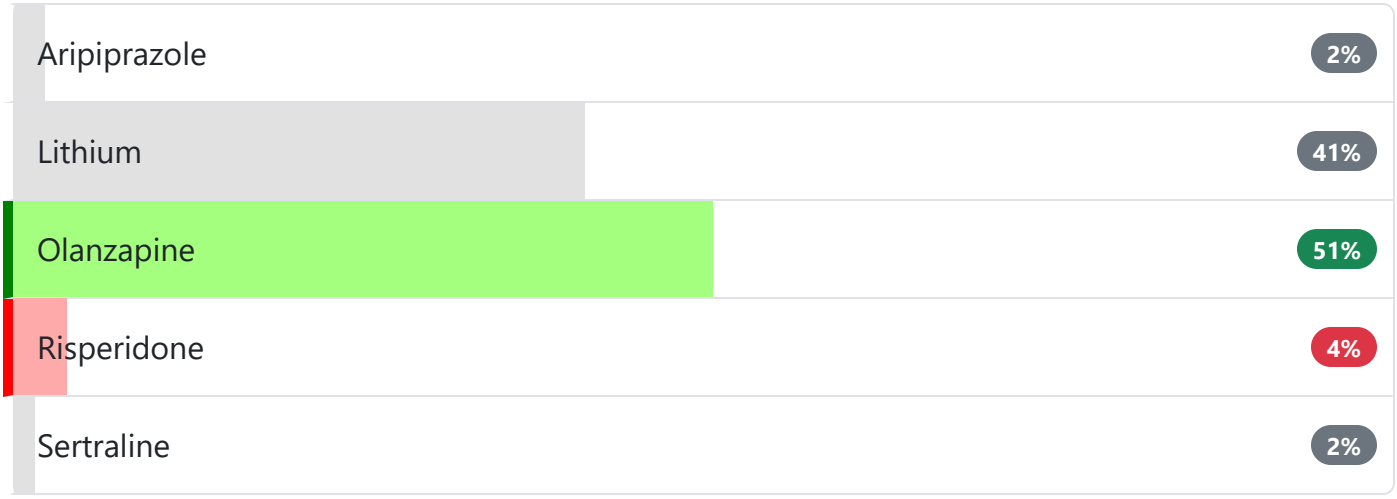

Long-term use of olanzapine is a significant risk factor for the development of type 2 diabetes mellitus

Important for me Less important

Although the pathophysiology is not known, it is recognised that **olanzapine** carries a significant risk for the development of type 2 diabetes mellitus, along with other antipsychotic agents. Olanzapine has an antagonistic effect on muscarinic acetylcholine receptor 3 which has been found to be crucial in glucose-induced insulin resistance. Olanzapine also leads to hyperglycaemia through its antagonistic effects on dopamine 2 receptors (increased appetite), H1 histamine receptors (insulin resistance) and 5-HT2A serotonin receptors (insulin resistance).

As with all antipsychotic medications, **aripiprazole** can cause hyperglycaemia, however, it carries a small risk, compared to olanzapine which carries significant risk.

Long-term use of **lithium** can increase blood glucose levels. However, it carries a risk of the

development of diabetes insipidus rather than diabetes mellitus.

Risperidone carries an intermediate risk of weight gain and the development of diabetes. The risk of diabetes development is higher with olanzapine.

According to the BNF, **sertraline** should be used with caution in people with known diabetes. This is due to its increased effect on insulin secretion thus increasing the risk of hypoglycaemia. Sertraline has not been shown to increase the risk of type 2 diabetes.

Discuss (4) | Improve

Next question ›

Drug-induced impaired glucose tolerance ★

Drugs which are known to cause impaired glucose tolerance include:

- thiazides, furosemide (less common)
- steroids
- tacrolimus, ciclosporin
- interferon-alpha
- nicotinic acid
- antipsychotics

Beta-blockers cause a slight impairment of glucose tolerance. They should also be used with caution in diabetics as they can interfere with the metabolic and autonomic responses to hypoglycaemia

AI + Q 123

Next question ›

B I A T↕ Textbooks

High-yield textbook

Extended textbook

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A 20-year-old man is brought to the Emergency Department by the Police with agitation and aggression. He is accompanied by a friend, who says they had been out celebrating his brother's birthday and that the patient may have 'snorted something'.

He recalls the patient complaining of intense nasal pain for some time after he allegedly took the substance, but it soon settled and he seemed in good spirits; talking enthusiastically about seeing vivid lights and waves emanating from the club's speakers. As the night went on, however, he became increasing disturbed; shouting at other clubgoers and complaining about hearing threatening voices.

On examination, his temperature is 39.2ºC. His pulse is 117bpm and his blood pressure is 181/98mmHg. He is difficult to examine due to intermittent aggression, and he has lashed out on several occasions.

In order to ensure his own safety, a decision to sedate the patient is made. The patient is successfully restrained and cannulated but suffers a tonic-clonic seizure shortly afterward. He is given 4mg IV lorazepam, but then suffers a cardiac arrest from which he cannot be resuscitated.

Which drug is most likely to be responsible?

- Lysergic acid diethylamide (LSD)
- Nexus (2CB)
- Spice
- Gamma-hydroxybutyric acid (GHB)
- Methoxetamine

Submit answer

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A 20-year-old man is brought to the Emergency Department by the Police with agitation and aggression. He is accompanied by a friend, who says they had been out celebrating his brother's birthday and that the patient may have 'snorted something'.

He recalls the patient complaining of intense nasal pain for some time after he allegedly took the substance, but it soon settled and he seemed in good spirits; talking enthusiastically about seeing vivid lights and waves emanating from the club's speakers. As the night went on, however, he became increasing disturbed; shouting at other clubgoers and complaining about hearing threatening voices.

On examination, his temperature is 39.2ºC. His pulse is 117bpm and his blood pressure is 181/98mmHg. He is difficult to examine due to intermittent aggression, and he has lashed out on several occasions.

In order to ensure his own safety, a decision to sedate the patient is made. The patient is successfully restrained and cannulated but suffers a tonic-clonic seizure shortly afterward. He is given 4mg IV lorazepam, but then suffers a cardiac arrest from which he cannot be resuscitated.

Which drug is most likely to be responsible?

Lysergic acid diethylamide (LSD)	40%
Nexus (2CB)	19%
Spice	9%
Gamma-hydroxybutyric acid (GHB)	13%
Methoxetamine	19%

Nexus is a designer drug belonging to the 2C family; a group of ring-substituted phenethylamines similar in structure to 3,4-methylenedioxy-N-methylamphetamine (MDMA). 2C refers to the presence of two carbon atoms between the amine group and the benzene ring in the chemical structure.

Common features of 2C intoxication include agitation, aggression, and hallucinations, often complicated by the presence of serotonergic or sympathomimetic toxidromes. It is possible to

increase the hallucinogenic effects of these drugs by placing substituents on the aromatic ring at positions 2, 4, or 5. Synthesis of 2C drugs by amateur chemists can, therefore, lead to the inadvertent formation of dangerously potent agents.

2Cs are ingested orally or by insufflation, with the latter being extremely painful. At low doses, 2Cs have stimulating effects and cause increased visual, auditory and tactile sensations. Hallucinations occur at moderate doses, whilst high doses can cause frightening hallucinations, hypertension, tachycardia, hyperthermia, seizures and excited delirium.

Patients with excited delirium often exhibit aggression, hyperactivity, hyperthermia and, as in this case, unexpected cardiac arrest. It is thought to relate to excessive dopamine release in the mesolimbic pathways and has been linked to a number of 2C-related deaths.

LSD is also a hallucinogen, but it would not cause the hypertension or hyperthermia evident in the vignette. Fatal overdose is rare.

GHB is a CNS depressant that causes euphoria followed by coma. It has a short duration of action and toxicity is often maximal at presentation.

Methoxetamine is a ketamine derivative that causes dissociative symptoms, tachycardia, hypertension, confusion, and occasionally an acute cerebellar syndrome. It does not typically cause hallucinations.

Spice is a synthetic cannabinoid. Like marijuana, spice is usually smoked or ingested orally. Intoxication results in elevated mood, relaxation, and altered perception. Spice, however, is more likely than marijuana to cause hypertension, tachycardia, seizures and acute psychosis.

Discuss (23) | Improve

Next question >

Novel psychoactive substances

Novel psychoactive substances is the medical term for the many new substances which are chemically related to established recreational drugs such as MDMA and cannabis. They are often referred to as 'legal highs' although this is a misnomer in the UK, as their distribution and sale have been illegal since 2016.

The information below describes some of the common types:

Stimulants

- similar to MDMA, amphetamines and cocaine, resulting in increased levels of serotonin, dopamine and noradrenaline, resulting in a 'high' and feeling of euphoria
- a common example is a stimulant NPS is mephedrone ('bath salts','M-CAT'.'meow meow'). It is a cathinone and structurally similar to khat, a plant found in East Africa
- another example is benzylpiperazine ('Exodus', 'Legal X', 'Legal E')
- typically swallowed as a pill/powder ('bombing') or snorted
- adverse effect profile similar to MDMA/cocaine, with the risk of serotonin syndrome

Cannabinoids

- termed synthetic cannabinoid receptor agonists
- commonly referred to as 'spice'
- typically sprayed on to herbal mixtures which are then smoked. Also available in liquid form which is then inhaled using e-cigarettes
- similar adverse effects to cannabis

Hallucinogenics

- can be either dissociatives and psychedelics
- dissociatives produce a similar effect to ketamine, with a sense of not being connected to the physical body or time. A common dissociative NPS is methoxetamine ('mexxy')
- psychedelics have a similar effect to LSD although NPS versions may also be a stimulant

Depressant

- can be either opioid or benzodiazepine-based
- usually taken as a pill or a powder
- often structurally very similar to the original drug class, hence the adverse effects are similar
- benzodiazepine NPS often have a significantly longer half-life

Other substances include:

- Gamma-hydroxybutyric acid (GHB) and gamma-butyrolactone (GBL): 'G', 'Geebs' or 'Liquid Ecstasy'
- GHB causes respiratory depression. When taken with other respiratory depressants, most commonly alcohol, this can be potentially life threatening
- Nitrous oxide: 'Hippie crack'

For a more detailed overview please see the excellent review in BMJ 2017;356:i6848

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 74-year-old man presents increasing ankle swelling but is otherwise well. The swelling is uncomfortable and impacting on his quality of life. He takes amlodipine 10mg once daily (OD) and losartan 75mg OD. His blood pressure is 128/73 mmHg in clinic. His examination reveals pitting oedema in the ankles but nil else. He had a recent chest radiograph which was normal and an echocardiogram that showed good systolic function. What action would you take?

- Add furosemide 20mg once daily
- Add spironolactone 25mg once daily
- Fluid restrict to 1.5 litres a day
- Reduce amlodipine to 5mg once daily
- Prescribe compression stockings

Submit answer

Reference ranges

Score: **0%**

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Add furosemide 20mg once daily	4%
Add spironolactone 25mg once daily	2%
Fluid restrict to 1.5 litres a day	2%
Reduce amlodipine to 5mg once daily	86%
Prescribe compression stockings	6%

This is likely a medication side effect given his normal echocardiogram. Amlodipine is heavily associated with ankle swelling. Therefore its dose should be reduced and if the blood pressure rises a further agent can be added.

There is no indication for diuretic therapy at this stage since there is no heart failure and the blood pressure is controlled.

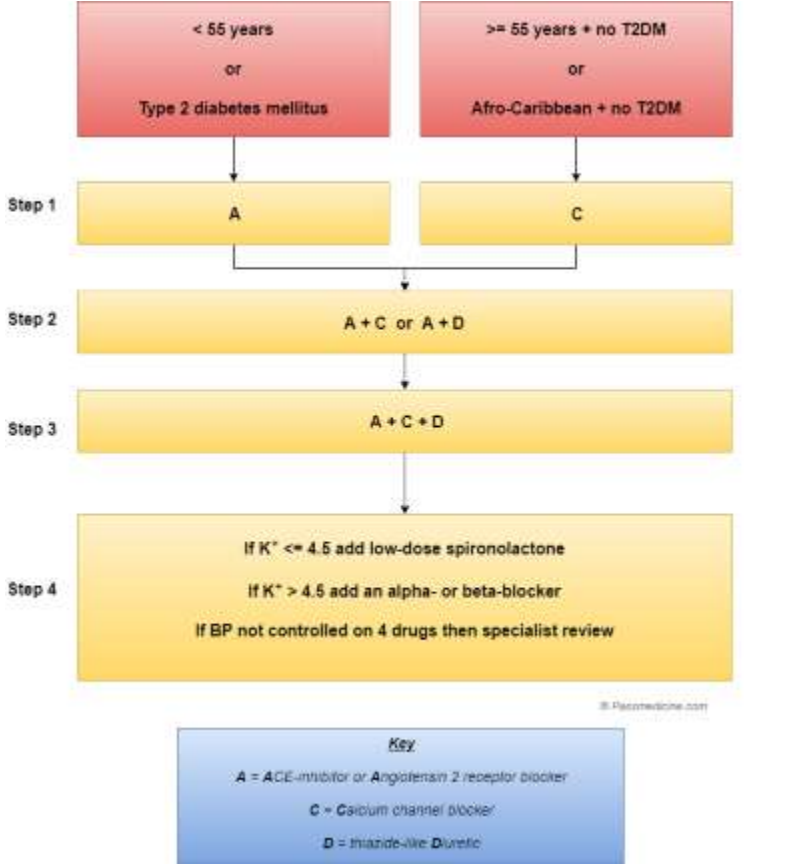
Discuss (4) | Improve

Next question

Calcium channel blockers

Calcium channel blockers are primarily used in the management of cardiovascular disease. Voltage-gated calcium channels are present in myocardial cells, cells of the conduction system and those of the vascular smooth muscle. The various types of calcium channel blockers have varying effects on these three areas and it is therefore important to differentiate their uses and actions.

Examples	Indications & notes	Side-effects and cautions
Verapamil	Angina, hypertension, arrhythmias Highly negatively inotropic Should not be given with beta-blockers as may cause heart block	Heart failure, constipation, hypotension, bradycardia, flushing
Diltiazem	Angina, hypertension Less negatively inotropic than verapamil but caution should still be exercised when patients have heart failure or are taking beta-blockers	Hypotension, bradycardia, heart failure, ankle swelling
Nifedipine, amlodipine, felodipine (dihydropyridines)	Hypertension, angina, Raynaud's Affects the peripheral vascular smooth muscle more than the myocardium and therefore do not result in worsening of heart failure but may therefore cause ankle swelling Shorter acting dihydropyridines (e.g. nifedipine) cause peripheral vasodilation which may result in reflex tachycardia	Flushing, headache, ankle swelling



Flow chart showing the management of hypertension as per current NICE guideliness

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

16 10

Calcium channel blockers

Suggest link Report broken link

Score: **15.1%**

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A 19-year-old female is admitted to the ward with reduced conscious level. Her mother found her at home lying on her bedroom floor. She has a past medical history of anxiety, depression, and hypothyroidism. On examination her heart rate is 110 beats per minute, blood pressure 124/80 mmHg, and respiratory rate is 36 breaths per minute.

Blood results are as follows:

Hb	122 g/l	Na^+	134 mmol/l
Platelets	488 * 10^9/l	K^+	5.6 mmol/l
WBC	14.2 * 10^9/l	Urea	10.8 mmol/l
Neuts	11.8 * 10^9/l	Creatinine	158 µmol/l
Lymphs	1.8 * 10^9/l	CRP	42 mg/l
Chloride	105 mmol/l		

An arterial blood gas is as follows:

PaO2	10.1 kPa
PCO2	2.2 kPa
pH	7.28
HCO3	12 mmol/l

What is the most likely diagnosis?

- Addisonian crisis
- Intracerebral hemorrhage
- Morphine overdose
- Encephalitis
- Salicylate overdose

Submit answer

Reference ranges

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Chloride	105 mmol/l		

An arterial blood gas is as follows:

PaO2	10.1 kPa
PCO2	2.2 kPa
pH	7.28
HCO3	12 mmol/l

What is the most likely diagnosis?

Addisonian crisis	7%
Intracerebral hemorrhage	1%
Morphine overdose	2%
Encephalitis	1%
Salicylate overdose	90%

Salicylate overdose can cause a mixed primary respiratory alkalosis and metabolic acidosis

Important for me Less important

The patient clearly has a metabolic acidosis as demonstrated by the low pH and low bicarbonate. With a primary metabolic acidosis we would expect the body to compensate by generating a respiratory alkalosis. Indeed this patient has hyperventilation and a low PCO2 indicating respiratory alkalosis. However it is important to remember that there is a limit for respiratory compensation. Even though respiratory compensation occurs relatively quickly, it can take up to twelve hours to reach maximal capacity.

The maximal respiratory compensation for metabolic disturbance can be calculated with:

- pCO2 (maximal response in kPa) = (0.2 * HCO3) + 1

If the patient's pCO2 lies within 0.5 kPa of this calculated value, then the respiratory response is appropriate. If the pCO2 is higher then either the compensation is in a very early stage or the patient has a superimposed respiratory acidosis. In this case we would expect a maximal compensation of 3.4 kPa. The pCO2 value is actually less than this indicating we also have a superadded primary metabolic alkalosis.

When dealing with a metabolic acidosis it is also important to work out the anion gap which is very useful for identifying the cause. In this case the anion gap = (Na + K) - (Cl +HCO3) = 22.6. This is a high anion gap metabolic acidosis (HAGMA). Causes of HAGMA include ketoacidosis, uraemia, lactic acidosis and toxins (e.g. salicylates, ethylene glycol and methanol).

The combination of HAGMA with a superadded primary respiratory alkalosis makes salicylate overdose the most likely diagnosis. Salicylates toxic effects on the brainstem result in hyperventilation and respiratory alkalosis.

Encephalitis can also affect the brainstem resulting in abnormal respiratory patterns and respiratory acid base balance disorders. However we would not expect there to be a severe HAGMA as evident in this case.

Morphine overdose typically causes respiratory depression resulting in a respiratory acidosis.

An Addisonian crisis typically causes a normal anion gap metabolic acidosis (NAGMA) and would not be associated with a primary respiratory alkalosis.

Discuss (8) Improve

Next question

Salicylate overdose

A key concept for the exam is to understand that salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis. In children metabolic acidosis tends to predominate.

Features

- hyperventilation (centrally stimulates respiration)
- tinnitus
- lethargy
- sweating, pyrexia*
- nausea/vomiting
- hyperglycaemia and hypoglycaemia
- seizures
- coma

Treatment

- general (ABC, charcoal)
- urinary alkalinization with intravenous sodium bicarbonate - enhances elimination of aspirin in the urine
- haemodialysis

Indications for haemodialysis in salicylate overdose

- serum concentration > 700mg/L
- metabolic acidosis resistant to treatment
- acute renal failure
- pulmonary oedema
- seizures
- coma

*salicylates cause the uncoupling of oxidative phosphorylation leading to decreased adenosine triphosphate production, increased oxygen consumption and increased carbon dioxide and heat production

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Medscape

2 0

Salicylate Toxicity review

Life in The Fast Lane

1 0

Salicylate toxicity

Suggest link

Report broken link

Score: **15.1%**

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You receive a request for a second opinion from a GP who has performed a general physical check on one of their new patients. He is a 54-year-old man who is from Thailand. He had been complaining of low mood, fatigue and sensitivity to the cold. His body mass index is 31 kg/m^2. The following is a list of investigations performed by the GP.

Na^+	141 mmol/l
K^+	4.8 mmol/l
Urea	9.8 mmol/l
Creatinine	142 µmol/l
CRP	4 mg/l

Bilirubin	14 µmol/l
ALP	86 u/l
ALT	27 u/l
Calcium	2.89 mmol/l
Albumin	39 g/l
TSH	24.0 mU/l
Free T4	0.8 pmol/l
Free T3	0.4 pmol/l

ECG: Heart rate 68, sinus rhythm, QRS width 128ms, flattened T waves V1 to V6

The patient has told the GP that he takes one medication regularly but is unable to give the name. Which medication is most likely to cause the following abnormalities?

- Bendroflumethiazide
- Amiodarone
- Lithium
- Carbimazole

Propylthiouracil

Submit answer

Reference ranges

Score: **0%**

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ECG: Heart rate 68, sinus rhythm, QRS width 128ms, flattened T waves V1 to V6

The patient has told the GP that he takes one medication regularly but is unable to give the name. Which medication is most likely to cause the following abnormalities?

Bendroflumethiazide	6%
Amiodarone	67%
Lithium	22%
Carbimazole	5%

Propylthiouracil 1%

The answer is Lithium. Long-term treatments with Lithium causes hypothyroidism in up to a third of patients with middle-aged females being the most likely demographic to get this complication. In addition, Lithium therapy is known to cause hypercalcaemia and hyperparathyroidism. Long-term lithium therapy is well known to cause renal impairment and weight gain on Lithium is common too. Cardiac side effects are rare on lithium but the most common ECG changes are flattened T waves and a widened QRS complex, in particular, a modified Brugada pattern may be seen.

Options D and E can cause hypothyroidism but are not known to cause any of the other abnormalities seen. Option A can cause weight gain and hypercalcaemia but is not known to cause any of the other abnormalities seen. Option B can cause hypothyroidism and similar ECG changes as seen here but is not known to cause any of the other abnormalities.

Discuss (7) Improve

Next question

Lithium toxicity

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure
- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- hyperreflexia
- acute confusion
- polyuria
- seizure

- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this
  - by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 23-year-old female student presents to the emergency department with a headache which has developed since starting her third year at university studying economics. She has also been noticing nausea and breathlessness. She had spent the summer travelling to Argentina and teaching English, after which she returned to the UK and moved into shared student accommodation in preparation for her next term. She had been well during her travels except for four days of diarrhoea shortly after she arrived. She has a past medical history of chlamydia which was treated by a local GUM clinic. She does not smoke and has minimal alcohol intake. On examination, she appears tired but otherwise well. Auscultation of her chest is normal. A urinary pregnancy test is positive.

Blood tests:

Hb	127 g/l
Platelets	336 * 10^9/l
WBC	9.4 * 10^9/l
Na^+	137 mmol/l
K^+	4.2 mmol/l
Urea	3.7 mmol/l
Creatinine	63 µmol/l

Arterial blood gas:

pH	7.41 g/l
pCO_2	3.6 mmHg
pO_2	9.4 mmHg
Na^+	137 mmol/l
FCOHb	22%

She is started on 15L of oxygen via a non-rebreather mask. How should she be further managed?

- Wean off oxygen guided by standard pulse oximetry saturation levels
- Add IV mannitol
- Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%

- [ ] Refer urgently for hyperbaric oxygen treatment
- [ ] Arrange urgently for non-invasive ventilation

Submit answer

Reference ranges

Score: **0%**

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Blood tests:

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Creatinine	63 µmol/l

Arterial blood gas:

pH	7.41 g/l
pCO_2	3.6 mmHg
pO_2	9.4 mmHg
Na^+	137 mmol/l
FCOHb	22%

She is started on 15L of oxygen via a non-rebreather mask. How should she be further managed?

Wean off oxygen guided by standard pulse oximetry saturation levels	2%
Add IV mannitol	1%
Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%	55%

Refer urgently for hyperbaric oxygen treatment 40%

Arrange urgently for non-invasive ventilation 2%

This is a case of carbon monoxide poisoning in a pregnant patient and therefore needs urgent consideration of hyperbaric oxygen treatment. The danger of carbon monoxide poisoning to the foetus is significant as carbon monoxide has greater affinity to foetal heamaglobin and the placenta is unable to substantially compensate for reduced oxygen delivery. Whilst mild to moderate poisoning has not been shown to cause any adverse outcomes, severe maternal poisoning has been shown to cause neurobehavioural deficits when treated with normobaric oxygen alone.

Standard pulse oximetry would be unable to assess oxygenation levels in carbon monoxide poisoning. Whilst if this patient was not pregnant, there would be no indication for hyperbaric oxygen treatment and maintaining high flow oxygen until asymptomatic or carbon monoxide levels would be less than 10% would then be appropriate. Non-invasive ventilation has no role in carbon monoxide poisoning, and mannitol is only indicated in cerebral oedema.

Discuss (7) Improve

Next question >

Carbon monoxide poisoning

Carbon monoxide has a high affinity for haemoglobin and myoglobin resulting in a left-shift of the oxygen dissociation curve and tissue hypoxia. There are approximately 50 per year deaths from accidental carbon monoxide poisoning in the UK.

Pathophysiology

- carbon monoxide binds readily to haemoglobin, forming carboxyhaemoglobin → reduced oxygen-carrying capacity
- in carbon monoxide poisoning the oxygen saturation of haemoglobin decreases leading to an early plateau in the oxygen dissociation curve

Questions may hint at badly maintained housing e.g. student houses.

Features of carbon monoxide toxicity

- headache: 90% of cases

- nausea and vomiting: 50%
- vertigo: 50%
- confusion: 30%
- subjective weakness: 20%
- severe toxicity: 'pink' skin and mucosae, hyperpyrexia, arrhythmias, extrapyramidal features, coma, death

Investigations

- pulse oximetry may be falsely high due to similarities between oxyhaemoglobin and carboxyhaemoglobin
- therefore a venous or arterial blood gas should be taken
- typical carboxyhaemoglobin levels
  - < 3% non-smokers
  - < 10% smokers
  - 10 - 30% symptomatic: headache, vomiting
  - > 30% severe toxicity
- an ECG is a useful supplementary investgation to look for cardiac ischaemia

Management

- patients with suspected carbon monoxide poisoning should be assessed in the emergency department
- 100% high-flow oxygen via a non-rebreather mask
  - from a physiological perspective, this decreases the half-life of carboxyhemoglobin (COHb)
  - should be administered as soon as possible, with treatment continuing for a minimum of six hours
  - target oxygen saturations are 100%
  - treatment is generally continued until all symptoms have resolved, rather than monitoring CO levels
- hyperbaric oxygen
  - due to the small number of cases the evidence base is limited, but there is some evidence that long-term outcomes may be better than standard oxygen therapy for more severe cases
  - therefore, discussion with a specialist should be considered for more severe cases (e.g. levels > 25%)
  - in 2008, the Department of Health publication 'Recognising Carbon Monoxide Poisoning' also listed loss of consciousness at any point, neurological signs other than headache, myocardial ischaemia or arrhythmia and pregnancy as indications for hyperbaric oxygen

Next question

Textbooks

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Media

Carbon Monoxide Poisoning

Kloss and Bruce - YouTube 3 2

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Score: **15.1%**

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A 52-year-old male was brought to the emergency department by his wife after being found confused and unsteady whilst walking. He had been well that morning but his wife had found him confused about 2 hours prior to presenting in the emergency department. He had a background of alcohol excess and bipolar disorder, for which he was on lithium. His wife reported the patient had recently been low in mood, triggered by his recent job loss.

On examination, the patient was confused with a flushed complexion, slurred speech, horizontal nystagmus, and a marked ataxic gait. Observations were as follows:

- oxygen saturations 97% on room air
- heart rate 65 bpm
- blood pressure 129/84mmHg
- temperature 37.2ºC

Blood results were as follows:

Creatinine	110 µmol/L	(55 - 120 µmol/L)
Urea	7 mmol/L	(2.0 - 7.0 mmol/L)
Na^+	140 mmol/L	(135 - 145 mmol/L)
K^+	4 mmol/L	(3.5 - 5.0 mmol/L)
Cl^-	100 mmol/L	(96 - 106 mmol/L)
Bicarbonate	17 mmol/L	(22 - 29 mmol/L)
pH	7.24	(7.35 - 7.45)
Anion gap	27 mmol/L	(<16 mmol/L)
Glucose	5.0 mmol/L	(4.0 - 11.1 mmol/L)
Lactate	2.5 mmol/L	(<2.3 mmol/L)
Ethanol	7.2 mmol/L	(<0.1 mmol/L)
Osmolality	359 mOsm/kg	(285 - 295 mOsm/kg)
Osmolar gap	59 mOsm/kg	(<10 mOsm/kg)
Salicylate	negative	
Paracetamol	negative	
Lithium	pending	

What is the most likely cause of the patient's presentation?

- Lithium overdose
- Ethanol toxicity
- Ethylene glycol toxicity
- Wernicke's encephalopathy
- Cyanide toxicity

Submit answer

Reference ranges

Score: **0%**

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Osmolar gap	59 mOsm/kg	(<10 mOsm/kg)
Salicylate	negative	
Paracetamol	negative	
Lithium	pending	

What is the most likely cause of the patient's presentation?

Lithium overdose	9%
Ethanol toxicity	16%
Ethylene glycol toxicity	65%
Wernicke's encephalopathy	9%
Cyanide toxicity	1%

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

This question is looking at the calculation of both the anion gap and osmolar gap.

This patient presents with features of intoxication. He is acidotic and has a raised anion gap as follows: (Na+K) - (HCO3+Cl) = 144 - 117 = 27mmol/L.
A normal anion gap is <16 mmol/L.

He also has a raised osmolar gap. This is calculated using the following equation:
Osmolar gap = measured osmolality - calculated osmolality.

- Measured osmolality = 359 mOsm/kg.
- Calculated osmolality = 2Na + glucose + urea = 288 + 7 + 5 = 300 mOsm/kg.
- Osmolar gap = 359 - 300 = 59 mOsm/kg.

A normal osmolar gap = <10 mOsm/kg.

We can add the ethanol concentration provided into our calculation of osmolality, to make it even more representative of the *true* osmolality. Despite this, we would still only have a calculated value of 307.2 mOsm/kg. This indicates that there is an unmeasured osmole present in the blood, causing a high osmolality, which is not explained by the presence of ethanol.

It is evident that this patient has consumed alcohol given the mildly raised serum ethanol concentration. Whilst ethanol toxicity can cause this presentation, the level is not high enough to account for such a severe presentation (according to Toxbase, symptoms of moderate toxicity such as ataxia and confusion typically occur when ethanol levels are 21.7 - 43.4 mmol/L). Furthermore, the high osmolar gap indicates the presence of another unmeasured osmole in the blood.

There are many causes of a high anion gap metabolic acidosis (please see the mnemonic

CATMUDPILES below for a helpful way to remember these!). One such cause is a high concentration of serum lactate. This patient has a mildly raised lactate which could be caused by cyanide poisoning (cyanide toxicity is typically associated with signs such as 'cherry red' skin and confusion). However, whilst lactate can cause a high *anion gap*, it does not cause a high *osmolar gap*. Furthermore, cyanide poisoning severe enough to cause this presentation would typically be associated with a much higher lactate concentration (a lactate level of >7mmol/L is typically associated with cyanide poisoning). Lactate levels are often falsely elevated in ethylene glycol poisoning.

Lithium overdose is not associated with a raised osmolar gap and typically presents with nausea, tremor, and muscle weakness (early signs).

Wernicke's encephalopathy can present with confusion, ataxia, nystagmus, and ophthalmoplegia, particularly in those with a history of alcohol excess. However, this is due to a deficiency in thiamine and does not explain the metabolic abnormalities present.

Interestingly, the treatment for ethylene glycol poising is fomepizole or ethanol. There have been cases where the co-ingestion of ethylene glycol and ethanol has provided some protective benefit, but ethanol levels much higher than 7mmol/L are normally required.

Some causes of a raised anion gap metabolic acidosis can be remembered by the mnemonic CATMUDPILES (from lifeinthefastlane.com):
- Cyanide, carbon monoxide
- Alcoholic ketoacidosis
- Toluene
- Methanol, metformin
- Uraemia
- Diabetic ketoacidosis
- Phenformin, pyroglutamic acid, paraldehyde, propylene glycol, paracetamol
- Iron, isoniazid
- Lactate (numerous causes)
- Ethanol, ethylene glycol
- Salicylates

Some causes of a high osmolar gap are also provided below (from lifeinthefastlane.com):
- Mannitol
- Methanol
- Ethylene glycol
- Sorbitol
- Polyethylene glycol (IV lorazepam)
- Propylene glycol (IV lorazepam, diazepam, and phenytoin)
- Glycine (TURP syndrome)
- Maltose (IV IG - Intragram)

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

Textbooks

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Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

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A 57-year-old male is admitted to hospital with an acute kidney injury (AKI). His past medical history includes polycystic kidney disease for which he received a renal transplant 4 years ago, and type 2 diabetes mellitus. His drug history includes tacrolimus, mycophenolate mofetil, amlodipine and simvastatin. Tacrolimus toxicity is suspected to have caused his AKI.

He informs you that he has only recently been discharged, where he was started on rifampicin and erythromycin for Legionnaires' disease. He was also started on pioglitazone after a diabetic team review.

Which of his other medications is the most likely culprit of the tacrolimus toxicity?

- Pioglitazone
- Erythromycin
- Simvastatin
- Rifampicin
- Mycophenolate mofetil

Submit answer

Reference ranges

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He informs you that he has only recently been discharged, where he was started on rifampicin and erythromycin for Legionnaires' disease. He was also started on pioglitazone after a diabetic team review.

Which of his other medications is the most likely culprit of the tacrolimus toxicity?

Pioglitazone	4%
Erythromycin	65%
Simvastatin	5%
Rifampicin	24%
Mycophenolate mofetil	3%

Despite his complicated medical and drug history, this is a relatively simple question about drug interactions.

Erythromycin is a potent inhibitor of the cytochrome P450 system, which means it can have a rapid effect on levels of other drugs metabolised by this system, e.g. tacrolimus, warfarin.

Clarithromycin, fluconazole and clotrimazole are also inhibitors.

Rifampicin is an enzyme inducer, which would cause decreased tacrolimus concentrations, potentially leading to symptoms of organ rejection.

P450 enzyme system

Induction usually requires prolonged exposure to the inducing drug, as opposed to P450 inhibitors, where effects are often seen rapidly

Inducers of the P450 system include

- antiepileptics: phenytoin, carbamazepine
- barbiturates: phenobarbitone
- rifampicin
- St John's Wort
- chronic alcohol intake
- griseofulvin
- smoking (affects CYP1A2, reason why smokers require more aminophylline)

Inhibitors of the P450 system include

- antibiotics: ciprofloxacin, erythromycin
- isoniazid
- cimetidine,omeprazole
- amiodarone
- allopurinol
- imidazoles: ketoconazole, fluconazole
- SSRIs: fluoxetine, sertraline
- ritonavir
- sodium valproate
- acute alcohol intake
- quinupristin

AI + Q 123

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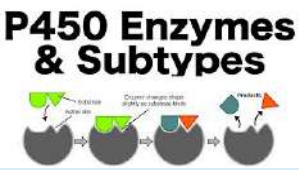

P450 Enzyme System (Inducers, Inhibitors, & Subtypes)

Dirty Medicine - YouTube 1 0

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A 23-year-old woman is reviewed following admission for paracetamol overdose. She ingested 25 grams of paracetamol and was started on N-acetylcysteine as her paracetamol level was 812 micromol/L eight hours following ingestion. She feels well, denying any abdominal pain or other symptoms. At what point should N-acetylcysteine be stopped?

- When INR <1.3 and ALT less than 2x upper limit
- When abdominal pain resolved
- When pH is >7.35
- When paracetamol levels become undetectable
- After 48 hours

Submit answer

Reference ranges

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When INR <1.3 and ALT less than 2x upper limit	44%
When abdominal pain resolved	2%
When pH is >7.35	15%
When paracetamol levels become undetectable	24%
After 48 hours	15%

The correct answer is when INR <1.3 and ALT less than 2x upper limit. Note that the primary measure of synthetic liver function is clotting which is why it is so closely monitored in paracetamol overdose and helps indicate when the liver is improving. pH is a useful measure for criteria for a liver transplant but is not helpful in most cases as it generally remains normal despite severe liver dysfunction. Abdominal pain is often present but not always and is therefore not a very sensitive marker. Paracetamol levels do not reflect the extent of liver damage and they can be very difficult to interpret in staggered overdoses. Depending on the extent of liver damage, treatment may need to be longer than 48hrs and in some cases shorter.

Discuss (6) | Improve

Next question

Paracetamol overdose: management

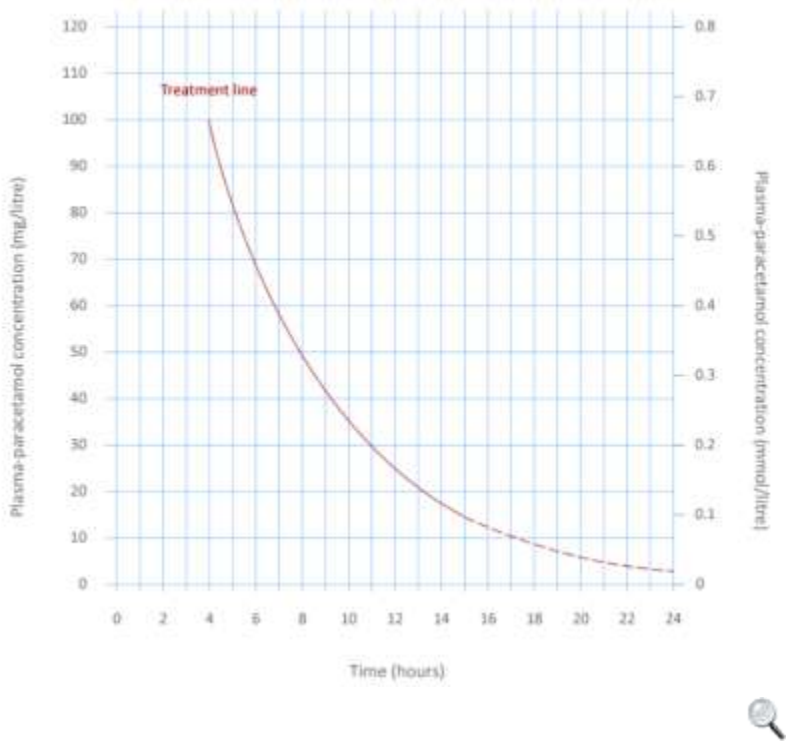
The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

Next question

Textbooks

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Links

Medicines and Healthcare products Regulatory Agency

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2012 Paracetamol overdose update

Suggest link Report broken link

Score: **15.1%**

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A 62-year-old man is currently being treated with linezolid for MRSA bacteraemia. Which of the following medications may need to be discontinued whilst this is being administered?

- Metformin
- Citalopram
- Alendronic acid
- Candesartan
- Atorvastatin

Submit answer

Reference ranges

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A 62-year-old man is currently being treated with linezolid for MRSA bacteraemia. Which of the following medications may need to be discontinued whilst this is being administered?

Metformin	11%
Citalopram	44%
Alendronic acid	8%
Candesartan	6%
Atorvastatin	30%

Linezolid was originally discovered as a psychotropic agent with antidepressant effects through mild reversible nonselective inhibition of monoamine oxidase (MAO), before it was found to have antibiotic efficacy against drug-resistant gram-positive cocci. In combination with serotonin agonists there is a risk of serotonin syndrome. Therefore, when using linezolid it must be considered to discontinue SSRIs.

There are no known listed interactions between alendronic acid, candesartan and atorvastatin.

Linezolid may potentially enhance the hypoglycaemic effect of metformin. Therefore careful capillary glucose monitoring should occur in a patient on both medications, but this is not necessarily a reason to discontinue the drug.

Discuss (3) | Improve

Next question

Serotonin syndrome

Causes

- monoamine oxidase inhibitors
- SSRIs

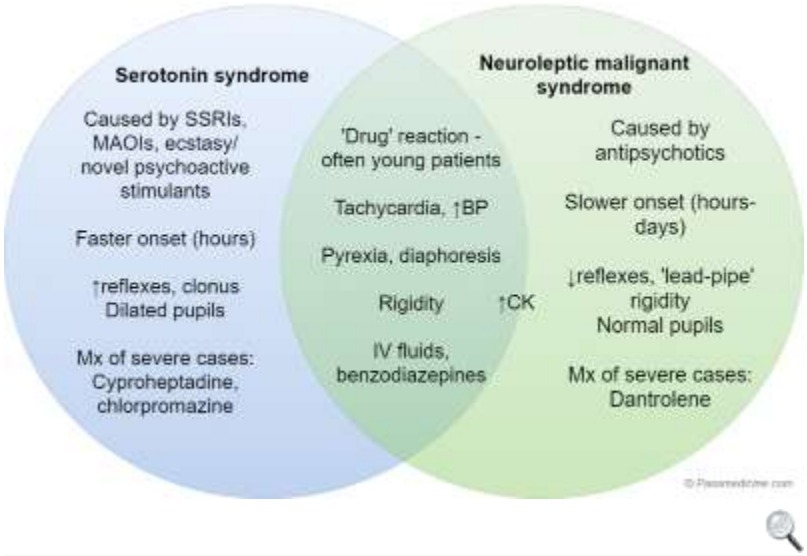
    - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
    - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

Features

- neuromuscular excitation
    - hyperreflexia
    - myoclonus
    - rigidity
- autonomic nervous system excitation
    - hyperthermia
    - sweating
- altered mental state
    - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

Next question

Textbooks

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Links

Medsafe 1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

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Score: **15.1%**

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A 72-year-old lady is admitted with a tonic-clonic seizure. She was initially treated with two doses of 10mg diazepam however she failed to respond. She was then prescribed a loading dose of intravenous phenytoin and then prescribed maintenance dose phenytoin. Past medical history included a previous stroke, gallstones and hypertension. Drug history included clopidogrel, amlodipine, simvastatin. She recovered well and was discharged 6 days later.

Three weeks after her hospital admission she visits her GP accompanied by her husband complaining of feeling generally unwell and a rash. On examination she has a wide spread morbilliform eruption on most of her body and some mild right upper quadrant pain. The GP could palpate cervical and inguinal lymph nodes. Observations heart rate 110 regular, blood pressure 120/80, saturations 97% on air and temperature 38.2. The GP sends the patient to hospital for further tests. Bloods show:

Hb	110 g/l
WCC	14 * 10^9/l
Neutrophils	7.2 * 10^9/l
Eosinophils	2.3 * 10^9/l
Platelets	81 * 10^9/l
Na^+	141 mmol/l
K^+	4.3 mmol/l
Creatinine	101 µmol/l
Urea	6.8 mmol/l
Bilirubin	22 µmol/l
Alk phos	80 u/l
ALT	150 u/l
Albumin	32 g/l
INR	1.1

What is the most likely diagnosis?

- Lymphoma
- DRESS syndrome

- Cholecystitis
- EBV
- Meningococcal septicaemia

Submit answer

Reference ranges

Score: **0%**

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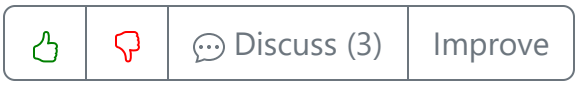
Alk phos	80 u/l
ALT	150 u/l
Albumin	32 g/l
INR	1.1

What is the most likely diagnosis?

Lymphoma	1%
DRESS syndrome	91%

Cholecystitis	1%
EBV	5%
Meningococcal septicaemia	2%

This patient has DRESS syndrome (drug reaction with eosinophilia and systemic symptoms) after starting phenytoin.



Next question

DRESS syndrome

DRESS is an unexpected, severe reaction to medication. Several organs are commonly affected including the skin, liver, kidneys, lungs and heart. Not all of these organs may be affected at any one time.

Classically patients develop a morbilliform skin rash in 80% cases which often leads to an exfoliative dermatitis, high fever, and inflammation of one or more organs. Vesicles and bullae may be seen. Erythroderma can occur in 10% of cases, mucosal involvement in 25% and facial swelling in 30%. The reaction usually occurs 2-8 weeks after commencing the offending drug.

Patients may develop systematic symptoms which can include haematological abnormalities (raised and low white count, eosinophilia (in 30% this is > $2.0 * 10^9$ /L, thrombocytopaenia, anaemia, atypical lymphocytes), enlarged lymph nodes (75%), 10% develop kidney disease which is usually mild (interstitial nephritis is common, renal failure is rare), myocarditis, pericarditis, liver enlargement, hepatitis and rarely hepatic necrosis with liver failure (abnormal liver function tests are found in 70-90% in particularly raised transaminases), lung disease (pneumonitis, pleuritis, pneumonia), neurological involvement which may lead to meningitis and encephalitis, gastrointestinal symptoms, in severe cases, acute colitis and pancreatitis can occur, and endocrine abnormalities may include thyroiditis and diabetes.

Diagnosis can be very difficult and it may also be difficult to determine the exact drug causing the hypersensitivity as first exposure may have started 8 weeks prior. Common drugs causing DRESS include allopurinol, anti-epileptics, antibiotics, immunosuppresants, HIV treatment and NSAIDS. A careful drug history should be taken.

The diagnosis should be suspected when there is presence of the triad of extensive skin rash, high

fever, and organ involvement, supported by a finding of eosinophilia and abnormal liver function tests. RegiSCAR has proposed a diagnostic criteria and patients require at least 3 of the following:

- Hospitalisation
- Reaction suspected to be drug related
- Acute skin rash
- Fever about 38ºC
- Enlarged lymph nodes at two sites
- Involvement of at least one internal organ
- Blood count abnormalities such as low platelets, raised eosinophils or abnormal lymphocyte count.

Skin biopsy can help to confirm the diagnosis. This may show inflammatory infiltrate in particularly eosinophils, extravasated erythrocytes and oedema. Regular blood tests including FBC, clotting, liver and renal function, CK, viral screen, glucose and thyroid function tests should be taken. Investigations looking for complications should be undertaken including ECG, CXR, echocardiogram, and urinalysis.

DRESS syndrome treatment requires all medications that are a possible culprits to be stopped and supportive care started. Antihistamines, topical steroids and emollients can be used to help control the rash. Careful fluid balance is necessary and clinicians should be aware of the patients nutritional status. Skin should be regularly checked and secondary infections should be treated with antibiotics. Systemic steroids may be started in severe cases where exfoliative dermatitis / pneumonitis / hepatitis is present. Occasionally immunosuppressants, intravenous immunoglobulin and plasmapheresis may be started. Potential culprit drugs should not be restarted again. The mortality is around 8%.

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media

DRESS Syndrome

Armando Hasudungan - YouTube 6 0

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Score: **15.1%**

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You are asked to review a 75-year-old lady on the surgical ward. She has malignant carcinoma of the colon and had a large bowel resection seven days ago. Despite being on dalteparin since admission, she had developed a right-sided deep venous thrombosis (DVT). She tells you she had a similar problem ten years ago and had to take 'blood-thinning injections' for several months.

On examination, she looks well with no signs of respiratory distress. Her oxygen saturations are 99% on room air and on auscultation she has vesicular breath sounds throughout both lung fields.

Her pre-operative blood tests are as following:

Hb	119 g/l	Na^+	135 mmol/l	Bilirubin	12 mol/l
Platelets	457 * 10^9/l	K^+	4.5 mmol/l	ALP	111 u/l
WBC	6.8 * 10^9/l	Urea	4.9 mmol/l	ALT	45 u/l
Neuts	4.0 * 10^9/l	Creatinine	81 µmol/l	γGT	30 u/l
Lymphs	0.9 * 10^9/l			Albumin	34 g/l
Eosin	0.0 * 10^9/l				

Her blood results today are as follows:

Hb	105 g/l	Na^+	133 mmol/l	Prothrombin time	10.7 s
Platelets	77* 10^9/l	K^+	4.1 mmol/l	APTT	27.5s
WBC	7.6 * 10^9/l	Urea	5.7 mmol/l	APTT ratio	45 u/l
Neuts	4.9 * 10^9/l	Creatinine	98 µmol/l	D-Dimer	>1000 ng/ml
Lymphs	1.1 * 10^9/l			Albumin	31 g/l
Eosin	0.1 * 10^9/l				

Which of the following represents the optimal management for this patient while she remains an inpatient?

- Warfarin
- Bivalirudin

- [ ] IV heparin infusion
- [ ] Plasma exchange
- [ ] Rivaroxaban

Submit answer

Reference ranges

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Lymphs	1.1 * 10^9/l			Albumin	31 g/l
Eosin	0.1 * 10^9/l				

Which of the following represents the optimal management for this patient while she remains an inpatient?

Warfarin	6%
Bivalirudin	54%

IV heparin infusion	14%
Plasma exchange	5%
Rivaroxaban	21%

This lady has heparin-induced thrombocytopenia (HIT). She needs anticoagulation but should ideally be switched to a non-heparin drug for now. Bivalirudin is a direct thrombin inhibitor and is not associated with HIT.

Plasma exchange would be the treatment of choice for thrombotic thrombocytopenic purpura, which is not the case here.

Warfarin causes a rapid fall in protein C levels, risking complications such as skin necrosis, so is not used acutely.

Discuss (7) Improve

Next question >

Heparin ★

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous

	Standard heparin	Low molecular weight heparin (LMWH)
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF

3 2

Heparin

British Society for Haematology

2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **15.1%**

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A 35-year-old man presents to the emergency department complaining of chest pain. He has no significant past medical history and does not take any regularly prescribed medications. He has been awake for twenty-four hours having ingested a large quantity of cocaine.

His observations are as follows: heart rate 111 beats per minute, respiratory rate 21/minute, blood pressure 180/100 mmHg, oxygen saturation 97% on room air and temperature 37ºC.

On examination, he is diaphoretic and anxious. Chest auscultation is normal. The heart sounds are normal with no added sounds or murmurs. The jugular venous pulse is not raised and there is no peripheral oedema. Abdominal and neurological examinations are normal.

ECG demonstrates sinus tachycardia with T wave inversion in leads V5-V6, I and AVL.

What medication should be avoided in this circumstance?

- Aspirin
- Benzodiazepines
- Bisoprolol
- Glyceryl trinitrate
- Sodium nitroprusside

Submit answer

Reference ranges

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On examination, he is diaphoretic and anxious. Chest auscultation is normal. The heart sounds are normal with no added sounds or murmurs. The jugular venous pulse is not raised and there is no peripheral oedema. Abdominal and neurological examinations are normal.

ECG demonstrates sinus tachycardia with T wave inversion in leads V5-V6, I and AVL.

What medication should be avoided in this circumstance?

Aspirin	4%
Benzodiazepines	9%
Bisoprolol	67%
Glyceryl trinitrate	10%
Sodium nitroprusside	9%

Cocaine toxicity - avoid beta-blockers

Important for me Less important

Bisoprolol is the correct answer. Beta-blockers should be avoided in cocaine-induced chest pain. The rationale is that beta-antagonism may result in unopposed alpha-mediated coronary vasospasm.

Aspirin is incorrect. This patient has an acute coronary syndrome and in addition to cocaine specific treatment, he will need antiplatelet treatment and coronary angiography.

Benzodiazepines are incorrect. This class of medication is used early in the treatment of cocaine-induced chest pain.

Glyceryl trinitrate is incorrect. This class of medication is used in the management of cocaine-induced chest pain.

Sodium nitroprusside is incorrect. The use of this may be indicated in those patients suffering from cocaine toxicity and persistent hypertension.

Discuss (2) | Improve

Next question

Cocaine

Cocaine is an alkaloid derived from the coca plant. It is widely used as a recreational stimulant. The price of cocaine has fallen sharply in the past decade resulting in cocaine toxicity becoming a much more frequent clinical problem. This increase has made cocaine a favourite topic of question writers.

Mechanism of action

- cocaine blocks the uptake of dopamine, noradrenaline and serotonin

Adverse effects

- cardiovascular
  - coronary artery spasm → myocardial ischaemia/infarction
  - both tachycardia and bradycardia may occur
  - hypertension
  - QRS widening and QT prolongation
  - aortic dissection
- neurological
  - seizures
  - mydriasis
  - hypertonia
  - hyperreflexia
- psychiatric effects
  - agitation
  - psychosis
  - hallucinations
- others
  - ischaemic colitis is recognised in patients following cocaine ingestion. This should be considered if patients complain of abdominal pain or rectal bleeding

    - hyperthermia
    - metabolic acidosis
    - rhabdomyolysis

Management of cocaine toxicity

- in general, benzodiazepines are generally first-line for most cocaine-related problems
- chest pain:
    - benzodiazepines + glyceryl trinitrate
    - if myocardial infarction develops then primary percutaneous coronary intervention
- hypertension: benzodiazepines + sodium nitroprusside
- the use of beta-blockers in cocaine-induced cardiovascular problems is a controversial issue
    - the American Heart Association issued a statement in 2008 warning against the use of beta-blockers (due to the risk of unopposed alpha-mediated coronary vasospasm) but many cardiologists since have questioned whether this is valid
    - if a reasonable alternative is given in an exam it is probably wise to choose it

Next question

Textbooks

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Media

Cocaine Dependence

Osmosis - YouTube 4 2

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Score: **15.1%**

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A 68-year-old man is admitted with a two day exacerbation of COPD. He arrives unwell and is started on steroids, nebulisers, aminophylline, ciprofloxacin and non invasive ventilation. He improves well and is weaned off the ventilation by his second day but starts to complain of palpitations and chest pain on the third day. He is usually well taking fostair twice a day. He used to smoke and drinks 25 units of alcohol a week.

You review him and find he has a pulse of 134 beats per minute that is irregular. He is clammy and his blood pressure is 96/76mmHg. He has a chest that is wheezy throughout and is saturating at 91% with 2 litres of nasal cannulae oxygen. He is visibly trembling and feels nauseous. A cardiac monitor is attached showing an irregular narrow complex tachycardia.

Na^+	145 mmol/l
K^+	3.2 mmol/l
Urea	4.5 mmol/l
Creatinine	76 µmol/l
Theophylline level	28 mcg/ml (normal range 10-20)
ECG	irregular narrow complex tachycardia with no visible p-waves
Chest x-ray	hyperexpanded film, no consolidation seen

What is the likely cause of his drug toxicity?

- Ciprofloxacin
- Hypokalaemia
- Anxiety
- Hidden pneumothorax
- Alcohol withdrawal

Submit answer

Reference ranges

Score: **0%**

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Theophylline level	28 mcg/ml (normal range 10-20)
ECG	irregular narrow complex tachycardia with no visible p-waves
Chest x-ray	hyperexpanded film, no consolidation seen

What is the likely cause of his drug toxicity?

Ciprofloxacin	73%
Hypokalaemia	17%
Anxiety	1%
Hidden pneumothorax	2%
Alcohol withdrawal	7%

Quinolones -> inhibit liver enzymes -> risk of theophylline toxicity in COPD/asthma exacerbation

Important for me Less important

This gentleman has developed new fast atrial fibrillation despite his initial improvement. The hypokalaemia probably contributes but it is clear he is toxic with aminophylline. Aminophylline concentrations are highly sensitive depending on the speed of liver metabolism. Ciprofloxacin is a liver enzyme inhibitor and when started alongside aminophylline, patients can be slow to metabolise and rapidly develop theophylline toxicity unless the dose is reduced. Alcohol withdrawal and anxiety are two differentials if no abnormalities showed on the investigations. A hidden pneumothorax is unlikely to be the cause here.

Discuss (2) Improve

Next question

Quinolones

Quinolones are a group of antibiotics which work by inhibiting DNA synthesis and are bactericidal in nature. Examples include:

- ciprofloxacin
- levofloxacin

Mechanism of action

- inhibit topoisomerase II (DNA gyrase) and topoisomerase IV

Mechanism of resistance

- mutations to DNA gyrase, efflux pumps which reduce intracellular quinolone concentration

Adverse effects

- lower seizure threshold in patients with epilepsy
- tendon damage (including rupture) - the risk is increased in patients also taking steroids
- cartilage damage has been demonstrated in animal models and for this reason quinolones are generally avoided (but not necessarily contraindicated) in children
- lengthens QT interval

Contraindications

- Quinolones should generally be avoided in women who are pregnant or breastfeeding
- avoid in G6PD

123

Next question

Textbooks

High-yield textbook

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Links

BNF

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Quinolones

Suggest link

Report broken link

Score: **15.1%**

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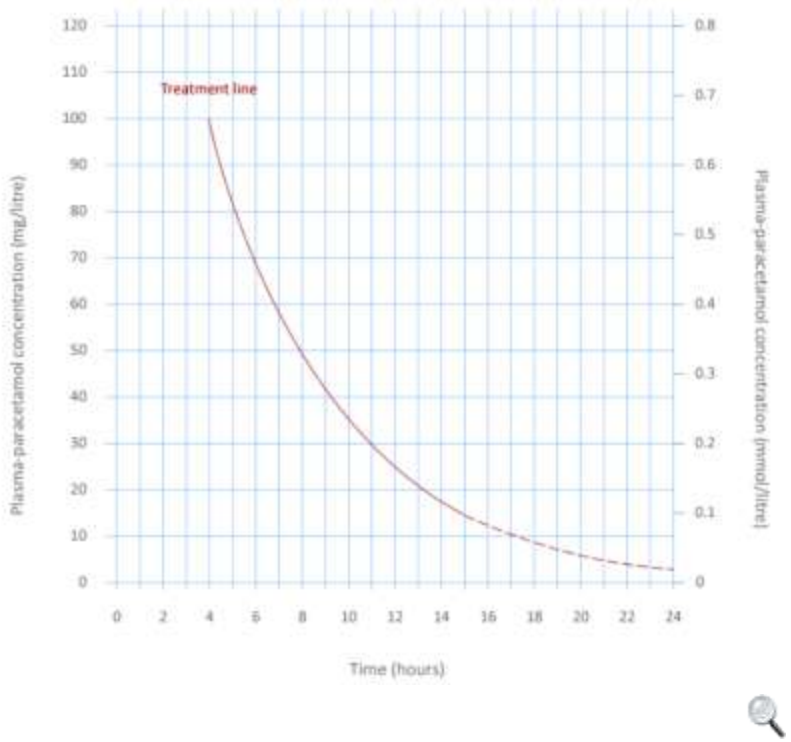
A 69-year-old woman attends the emergency department with a 2-day history of lower back pain after falling down the stairs. She has been taking 1g of paracetamol every 1-2 hours to control the pain but says this has not helped her symptoms. Over the last 8 hours, she now has developed right upper quadrant pain and nausea. Her last dose of paracetamol was 45 minutes ago.

Laboratory investigations:

Hb	123 g/L	(115 - 160)
Na^+	137 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	77 µmol/L	(55 - 120)
Bilirubin	16 µmol/L	(3 - 17)
ALP	99 u/L	(30 - 100)
ALT	42 u/L	(3 - 40)
Paracetamol levels	35 mg/L	(10-30)



What is the most initial step in the management of this patient?

- [ ] Activated charcoal
- [ ] Initiate N-acetylcysteine
- [ ] No treatment indicated
- [ ] Referral to liaison psychiatry
- [ ] Repeat paracetamol levels at 4 hours from last ingestion

Submit answer

Reference ranges

Score: **0%**

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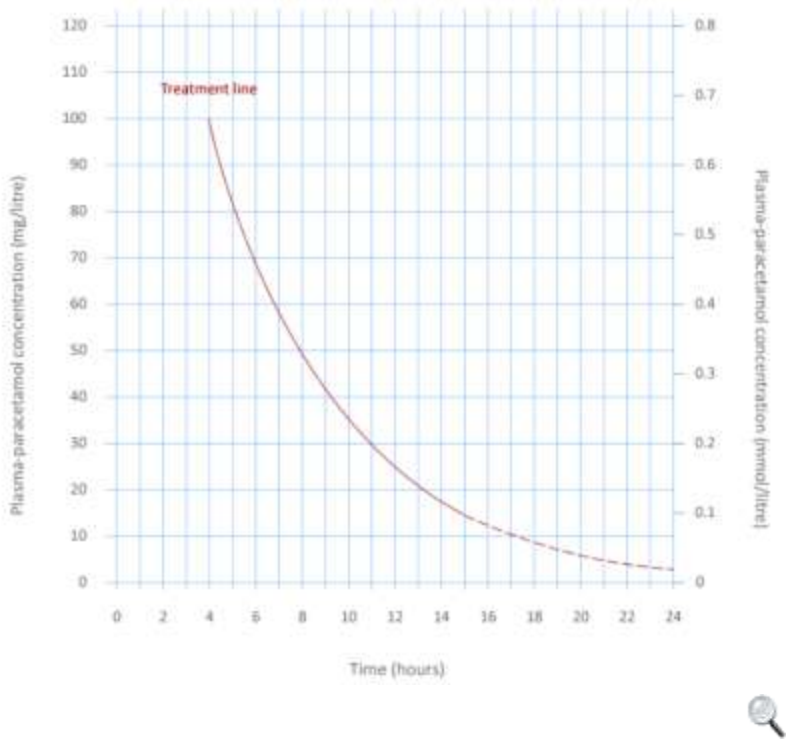
ALT	42 u/L	(3 - 40)
Paracetamol levels	35 mg/L	(10-30)



What is the most initial step in the management of this patient?

Activated charcoal	12%
Initiate N-acetylcysteine	70%
No treatment indicated	8%
Referral to liaison psychiatry	0%
Repeat paracetamol levels at 4 hours from last ingestion	10%

This patient has taken a staggered overdose of paracetamol, having taken 1g every 1-2 hours for back pain. Regardless of the paracetamol level and where it corresponds on the treatment graph, patients should be immediately treated with **N-acetylcysteine** to prevent liver toxicity.

Activated charcoal is incorrect. This can be administered within the 1st hour of paracetamol ingestion, to reduce absorption. However, in the event of taking a staggered overdose, NICE guidelines recommend commencing N-acetylcysteine regardless of the paracetamol levels and time following ingestion.

No treatment indicated is incorrect. Not treating a staggered paracetamol overdose is dangerous and could lead to long-term hepatic complications or death from acute liver failure.

Referral to liaison psychiatry is incorrect. Whilst patients who have presented with a drug overdose should be considered for referral to liaison psychiatry, this patient has no other presenting signs to suggest an intentional overdose. Nevertheless, the most important initial step is to administer treatment with N-acetylcysteine prior to referring to liaison psychiatry.

Repeat paracetamol levels at 4 hours from last ingestion is incorrect. In most cases of paracetamol overdose, guidelines state that paracetamol levels should be taken 4 hours from the last ingestion. If the paracetamol levels are then over the treatment line, then treatment with N-acetylcysteine can be commenced. However, the exception to the rule includes a large overdose (>150mg/kg) or a staggered overdose of paracetamol.

👍 👎 💬 Discuss (2) Improve

Next question ›

Paracetamol overdose: management ★

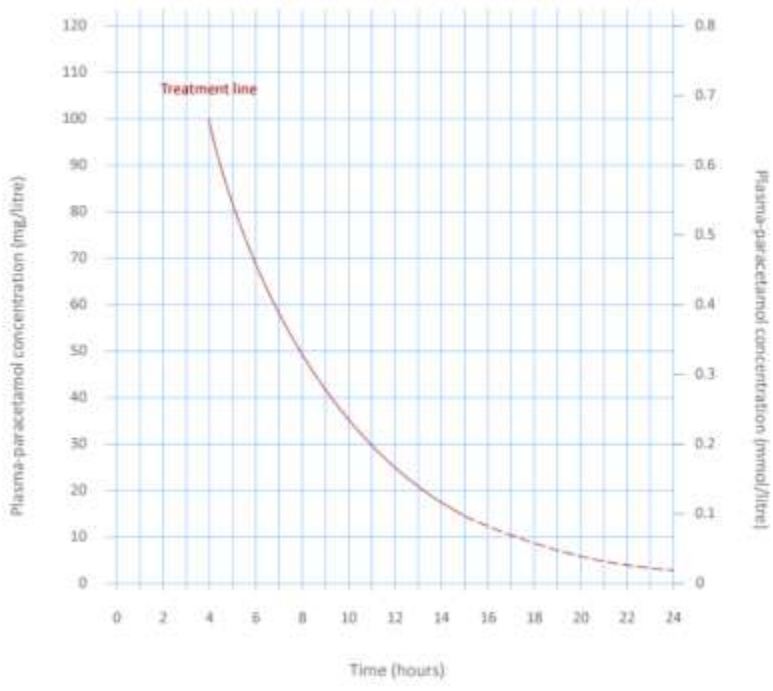
The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medicines and Healthcare products Regulatory Agency

3 3

2012 Paracetamol overdose update

Suggest link Report broken link

Score: **15.1%**

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A 28-year-old lady is brought in by her mother having taken an overdose of an unknown medication. She has recently broken up with her boyfriend and has been fired from her job for turning up late to work persistently. She was found in her room by her mother with a small bottle of vodka which was empty and some burnt medication packaging. She has no history of mental health disease, although did self-harm (arm cutting) twice whilst a teenager. She has no medical history other than being treated for malaria during a recent trip to west Africa. The patient is tearful, regrets the overdose and is currently complaining of nausea, epigastric discomfort, slightly blurred vision and a ringing in her ears.

On examination, her heart rate is 115 beats/min, her blood pressure 98/48 mmHg and she appears flushed. Her respiratory rate is 18 breaths per minute and her oxygen saturations 98% breathing room air. Her Glasgow coma score is 15/15 and her pupils are equal and reactive. Her blood results are as follows.

Na^+	135 mmol/l
K^+	4.3 mmol/l
Urea	5.1 mmol/l
Creatinine	86 µmol/l
Bilirubin	18 µmol/l
ALP	65 u/l
ALT	56 u/l

What is the most likely toxin?

- Quinine overdose
- Ibuprofen overdose
- Alcohol intoxication
- Paracetamol overdose
- Malarone overdose

Submit answer

Reference ranges

Score: **0%**

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ALP	65 u/l
ALT	56 u/l

What is the most likely toxin?

Quinine overdose	83%
Ibuprofen overdose	5%
Alcohol intoxication	3%
Paracetamol overdose	3%
Malarone overdose	6%

Cinchonism is the syndrome described (blurred vision, tinnitus, nausea...) and can be secondary to

quinine or quinidine ingestion as an overdose, but also less commonly with normal dosing. Although uncommon in the western world use of quinine as a drug in overdose is not uncommon in malaria endemic areas such as south-east Asia. The tinnitus may be associated with transient loss of high frequency hearing loss. The blurred vision can progress to complete blindness, which in some cases can be permanent. None of the other toxins would explain this toxidrome.

Discuss (4) Improve

Next question

Quinine toxicity (cinchonism)

Quinine is a remarkably toxic drug; something which is not so readily acknowledged. It is used as an antimalarial drug and also as a prophylactic agent against leg cramps, although both uses are increasingly falling from vogue due to the availability of better, safer agents. Quinine toxicity, known as cinchonism, may be fatal, usually by cardiac arrhythmia or flash pulmonary oedema in the short term, although incipient renal failure may be fatal more long-term.

Cardiac arrhythmia is a common finding in cinchonism due to blockade of sodium and potassium channels prolonging QRS and QT intervals respectively and these rhythms may degenerate into ventricular tachyarrhythmias or fibrillation causing death. **Hypoglycaemia** is also a common finding in cinchonism since quinine stimulates pancreatic insulin secretion and this should be corrected rapidly if present. Unlike in the above case, flash pulmonary oedema may develop causing hypoxia and necessitating positive pressure ventilation. Classical hallmarks of cinchonism are tinnitus, visual blurring, flushed and dry skin and abdominal pain.

Clinically, quinine toxicity is difficult to distinguish from aspirin poisoning and so measurement of serum salicylate levels is important when this clinical picture is seen. In terms of management however, whereas aspirin can be cleared from overdose victims by haemofiltration, quinine cannot be extracted easily by extracorporeal methods. Central nervous symptoms such as tinnitus, deafness and visual defects which may occur with aspirin are usually transient whereas quinine leaves permanent neural damage, if the patient survives.

Management of quinine poisoning is largely supportive with fluids, inotropes and bicarbonate as needed as well as positive pressure ventilation for pulmonary oedema.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 65-year-old woman attends the emergency department complaining of three weeks of gradual visual loss. She has a past medical history of rheumatoid arthritis, type 2 diabetes, and pulmonary tuberculosis. Her medications include methotrexate, hydroxychloroquine, metformin, rifampicin, ethambutol hydrochloride, pyrazinamide, isoniazid and pyridoxine.

On examination, visual acuity is 20/50 in both eyes. There are central scotomas bilaterally on visual field testing. The macula and fovea are normal.

Blood tests:

ESR 88 mm/hr	(<20)
Hba1c	54 mmol/mol (<48)

What is the likely cause of her presentation?

- Ethambutol treatment
- Giant cell arteritis
- Hydroxychloroquine treatment
- Multiple sclerosis
- Vitreous haemorrhage

Submit answer

Reference ranges

Score: **0%**

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Blood tests:

ESR 88 mm/hr	(<20)
Hba1c	54 mmol/mol (<48)

What is the likely cause of her presentation?

Ethambutol treatment	63%
Giant cell arteritis	4%
Hydroxychloroquine treatment	30%
Multiple sclerosis	2%
Vitreous haemorrhage	1%

Optic neuritis is common in patients taking ethambutol

Important for me Less important

Ethambutol treatment is correct. The patient presents with insidious onset painless loss of central vision. This is typical of optic neuropathy associated with ethambutol treatment. Eventually, the optic nerve may become visually abnormal but it may be normal early in the course of the disease. The raised ESR is likely due to either tuberculosis or rheumatoid.

Giant cell arteritis is incorrect. This can cause visual loss and a raised ESR. However, visual loss in giant cell arteritis (GCA) is typically unilateral and sudden rather than bilateral and progressive.

Hydroxychloroquine treatment is incorrect. This can cause painless bilateral loss of vision. However, we would expect macular oedema, which is absent here. Hydroxychloroquine (HCQ) retinopathy typically occurs when patients are treated with HCQ for extended periods of time i.e. > 5 years.

Multiple sclerosis is incorrect. This can cause optic neuritis. However, this is more likely to be painful and unilateral rather than painless and bilateral.

Vitreous haemorrhage is incorrect. This can occur as a complication of diabetes. However, it would typically be unilateral and cause sudden painless loss of vision. Additionally, blood may be visible on fundoscopic examination.

Discuss (1) Improve

Next question >

Tuberculosis: drug side-effects and mechanism of action

Rifampicin

- mechanism of action: inhibits bacterial DNA dependent RNA polymerase preventing transcription of DNA into mRNA
- potent liver enzyme inducer
- hepatitis, orange secretions
- flu-like symptoms

Isoniazid

- mechanism of action: inhibits mycolic acid synthesis
- peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
- hepatitis, agranulocytosis
- liver enzyme inhibitor

Pyrazinamide

- mechanism of action: converted by pyrazinamidase into pyrazinoic acid which in turn inhibits fatty acid synthase (FAS) I
- hyperuricaemia causing gout
- arthralgia, myalgia
- hepatitis

Ethambutol

- mechanism of action: inhibits the enzyme arabinosyl transferase which polymerizes arabinose into arabinan
- optic neuritis: check visual acuity before and during treatment
- dose needs adjusting in patients with renal impairment

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

0 0

2016 Tuberculosis guidelines

Suggest link Report broken link

Media

Tuberculosis

Osmosis - YouTube 0 0

Report broken media

Score: **15.1%**

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A 45-year-old woman presents to the emergency department with bilateral hand tremor, unsteady gait, and blurry vision. Her past medical history is significant for bipolar disorder, which has been under control for the last year on lithium. Recently, her general practitioner started her on amlodipine for essential hypertension. Additionally, she started taking paracetamol and oral ibuprofen daily over the last week for knee pain related to a recent fall.

On examination, a coarse tremor is noted in both hands. There is also nystagmus, dysdiadochokinesia, and past pointing.

What is the best next step in managing this patient?

- Increase lithium dose
- Start haemodialysis
- Stop amlodipine
- Stop ibuprofen
- Stop paracetamol

Submit answer

Reference ranges

Score: **0%**

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On examination, a coarse tremor is noted in both hands. There is also nystagmus, dysdiadochokinesia, and past pointing.

What is the best next step in managing this patient?

Increase lithium dose	1%
Start haemodialysis	37%
Stop amlodipine	5%
Stop ibuprofen	55%
Stop paracetamol	2%

Lithium toxicity can be precipitated by NSAIDs

Important for me Less important

The patient presents with classic symptoms of lithium toxicity related to recent use of ibuprofen. **Increasing the dose of lithium** would cause further deterioration of her symptoms. Lithium should be held, and lithium levels should be measured to guide further management.

Management of lithium toxicity is mainly supportive. It is essential to discontinue lithium and the drug that precipitated lithium toxicity (e.g., ibuprofen, bendroflumethiazide) and consider administration of intravenous fluids based on the patient's fluid balance status. **Haemodialysis** may be required in the context of severe symptoms, kidney dysfunction, and significantly raised lithium levels, but it is not indicated as a first-line treatment for this patient.

Amlodipine does not interact with lithium and, as such, it can be continued for the management of essential hypertension.

This patient presents with classic features of lithium toxicity. This has been precipitated by ibuprofen ingestion. Non-steroidal anti-inflammatory drugs and thiazide-type diuretics can increase lithium levels and, as such, should be avoided in patients on lithium. In this case, **ibuprofen should be discontinued** in the first instance.

Paracetamol does not interact with lithium, and as such, it can be continued for the management of essential hypertension.

Discuss (2) | Improve

Next question >

Lithium toxicity ★

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure
- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- hyperreflexia
- acute confusion
- polyuria
- seizure
- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this

- by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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Back to top

A 24-year-old man presents to the accident and emergency department with his girlfriend after collapsing in a nightclub at 0400hrs. It is now 0600hrs On examination he is V on the AVPU scale with dry mucous membranes, he has flushed skin and feels very warm to the touch. There is mildly increased muscle tone, myoclonic jerks and hyperreflexia globally but worse in the lower limbs. Pupils are dilated but respond to light equally and bilaterally. He is disorientated and agitated during the examination, thrashing about in the bed and occasionally lashing out at people

Heart rate 126/min, blood pressure 90/60 mmHg, respiratory rate 18/min, temperature = 40.3ºC, sats 97% on 10l of oxygen.

Na^+	136 mmol/l
K^+	3.9 mmol/l
Urea	2.3 mmol/l
Creatinine	77 µmol/l
Glucose	4.6 mmol/l
Creatinine kinase	12,000 U/l

Bilirubin	25 µmol/l
ALP	185 u/l
ALT	125 u/l
γGT	144 u/l
Albumin	40 g/l

CT Head: No abnormality detected.

Lumbar puncture reveals:

Opening pressure	15 cmCSF
Appearance	Clear
Glucose	3.5 mmol/l
Protein	0.3 g/l

White cells	4 / mm³

He has a past medical history of schizoaffective disorder. His girlfriend shows you his regular medications, the prescription has been unchanged for 1 year. Olanzapine 15mg and Sertraline 150mg. His girlfriend tells you that he took an unknown white powder whilst in the club

What is the most appropriate treatment plan?

- IV Ceftriaxone
- Stop Olanzapine and give dantrolene
- Stop sertraline and give IV lorazepam and consider cyproheptadine
- Give IV naloxone
- Stop olanzapine and sertraline and supportive therapy

Submit answer

Reference ranges

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A 24-year-old man presents to the accident and emergency department with his girlfriend after collapsing in a nightclub at 0400hrs. It is now 0600hrs On examination he is V on the AVPU scale with dry mucous membranes, he has flushed skin and feels very warm to the touch. There is mildly increased muscle tone, myoclonic jerks and hyperreflexia globally but worse in the lower limbs. Pupils are dilated but respond to light equally and bilaterally. He is disorientated and agitated during the examination, thrashing about in the bed and occasionally lashing out at people

Heart rate 126/min, blood pressure 90/60 mmHg, respiratory rate 18/min, temperature = 40.3ºC, sats 97% on 10l of oxygen.

Na^+	136 mmol/l
K^+	3.9 mmol/l
Urea	2.3 mmol/l
Creatinine	77 µmol/l
Glucose	4.6 mmol/l
Creatinine kinase	12,000 U/l

Bilirubin	25 µmol/l
ALP	185 u/l
ALT	125 u/l
γGT	144 u/l
Albumin	40 g/l

CT Head: No abnormality detected.

Lumbar puncture reveals:

Opening pressure	15 cmCSF
Appearance	Clear
Glucose	3.5 mmol/l
Protein	0.3 g/l

White cells	4 / mm³

He has a past medical history of schizoaffective disorder. His girlfriend shows you his regular medications, the prescription has been unchanged for 1 year. Olanzapine 15mg and Sertraline 150mg. His girlfriend tells you that he took an unknown white powder whilst in the club

What is the most appropriate treatment plan?

IV Ceftriaxone	1%
Stop Olanzapine and give dantrolene	20%
Stop sertraline and give IV lorazepam and consider cyproheptadine	72%
Give IV naloxone	2%
Stop olanzapine and sertraline and supportive therapy	6%

The diagnosis is MDMA intoxication which has led to serotonin syndrome and rhabdomyolysis. The treatment for serotonin syndrome is stopping any serotonergic agents, using benzodiazepines for agitation and consideration of the use of serotonin antagonists such as cyproheptadine if there is severe autonomic disturbance. MDMA intoxication can lead to serotonin syndrome, especially if used in conjunction with any drug which increased serotonin transport across the synapses eg SSRIs such as sertraline.

Serotonin syndrome presents with agitation, reduced consciousness, flushing of the skin, hyperthermia, hyperreflexia, myoclonus (worse in the lower limbs), tachycardia, hypertension or labile BP. Most cases of serotonin syndrome present within 6 hours of any change in medication or dose i.e in this case after the MDMA.

Neuroleptic malignant syndrome would present in a similar way and would be treated by option B.However NMS usually evolves over 1-3 days with extreme muscle rigidity being a characteristic feature. NMS is less likely when on a lower potency antipsychotic such as Olanzapine and less likely when the dose has been stable for a long period of time.

Heroin overdose would not present with these features. Dilated pupils essentially rule this out and heroin is not usually a white powder which makes option D less likely.

Although meningitis can present with fever and reduced consciousness the normal LP rules out this diagnosis and therefore option A is an inappropriate treatment

Option E is inappropriate as this gentleman is severely unwell with autonomic instability, severe

hyperthermia and significant rhabdomyolysis with renal impairment. Active management other than supportive treatment would be recommended for this individual if the diagnosis were NMS or serotonin syndrome

Discuss (13) | Improve

Next question

Ecstasy poisoning

Ecstasy (MDMA, 3,4-Methylenedioxymethamphetamine) use became popular in the 1990's during the emergence of dance music culture.

Clinical features

- neurological: agitation, anxiety, confusion, ataxia
- cardiovascular: tachycardia, hypertension
- hyponatraemia
  - this may result from either syndrome of inappropriate ADH secretion or excessive water consumption whilst taking MDMA
- hyperthermia
- rhabdomyolysis

Management

- supportive
- dantrolene may be used for hyperthermia if simple measures fail

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Annals of Clinical Psychiatry 0 2

Serotonin syndrome vs neuroleptic malignant syndrome: A contrast of causes, diagnoses, and management

Suggest link Report broken link

Score: **15.1%**

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A 65-year-old woman attends the clinic with shortness of breath. This has progressively worsened over the past 3 months. She also complains of swollen ankles. She has a past medical history of breast cancer which was treated with docetaxel, cyclophosphamide, trastuzumab, and radiotherapy. She also has rheumatoid arthritis for which she takes methotrexate.

On examination, you note bilateral pitting oedema to the mid calves and coarse crepitations to the mid zones of the lungs. Observations are as follows: heart rate 92 beats per minute, respiratory rate 18 breaths per minute, blood pressure 125/75 mmHg, SpO2 94% on air, and temperature 37.2ºC.

An echo is performed:

Echo	Biventricular failure; left ventricle ejection fraction 28%; no evidence of pericardial thickening

What is the most likely implicated therapeutic?

- Cyclophosphamide
- Docetaxel
- Methotrexate
- Radiotherapy
- Trastuzumab

Submit answer

Reference ranges

Score: **0%**

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An echo is performed:

Echo	Biventricular failure; left ventricle ejection fraction 28%; no evidence of pericardial thickening

What is the most likely implicated therapeutic?

Cyclophosphamide	3%
Docetaxel	35%
Methotrexate	3%
Radiotherapy	2%
Trastuzumab	56%

Trastuzumab (Herceptin) - cardiac toxicity is common

Important for me Less important

Trastuzumab is correct. Trastuzumab (Herceptin) is a monoclonal antibody directed against the HER2/neu receptor. Cardiotoxicity is a well-recognised complication and is more common when anthracyclines have also been used.

Cyclophosphamide is incorrect. Although higher doses of this drug may produce significant cardiac toxicity (including fatal hemorrhagic myocarditis), the incidence is much less so compared to trastuzumab.

Docetaxel is incorrect. Cardiac failure is a rare side effect of this agent.

Methotrexate is incorrect. Several clinical trials have shown that methotrexate is associated with improved endothelial function, slower atherosclerosis progression, decreased risk of major cardiovascular adverse events, and benefits on overall survival.

Radiotherapy is incorrect. Constrictive pericarditis is a late complication of radiation therapy. Patients typically present with signs and symptoms of heart failure, similar to other causes of constrictive pericarditis. The absence of pericardial thickening on echo makes this a less likely diagnosis.

Discuss (2) Improve

Next question

Trastuzumab

Trastuzumab (Herceptin) is a monoclonal antibody directed against the HER2/neu receptor. It is used mainly in metastatic breast cancer although some patients with early disease are now also given trastuzumab.

Adverse effects

- flu-like symptoms and diarrhoea are common
- cardiotoxicity
  - more common when anthracyclines have also been used
  - an echo is usually performed before starting treatment

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Biologics

9 4

Trastuzumab in the management of early and advanced stage breast cancer

Suggest link Report broken link

Score: **15.1%**

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A 65-year-old female presents to her GP with a four days history of non-itchy rashes on her face, neck and bilateral upper limbs. Her chest and abdomen are not affected. She does regular gardening. Her past medical history includes myocardial infarction and asthma. Further history reveals that last week she was seen in the cardiology outpatient clinic for arrhythmias and was started on a new medication. Which of the following drug is most likely casuing the patient's symptom?

- Digoxin
- Dabigatran
- Verapamil
- Amiodarone
- Flecainide

Submit answer

Reference ranges

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Digoxin	8%
Dabigatran	3%
Verapamil	7%
Amiodarone	76%
Flecainide	7%

The distribution of the rash suggests phototoxicity. Among the list of the medications, only amiodarone and flecainide are assocaited with phototoxicity. However, flecainide is contraindicated in patients with history of myocardial infarction.

Discuss (3) | Improve

Next question

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

Score: **15.1%**

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A 22-year-old male is 'blue-lighted' to the Emergency Department having been found collapsed at a party. On arrival, his GCS is 3/15 and he is immediately intubated and ventilated by the Emergency Physicians.

On examination he is afebrile. He is mildly bradycardic at 53bpm and his blood pressure is 109/69mmHg. His pupils measure 2mm bilaterally. IV access is obtained and 400 micrograms of naloxone are administered without effect. The on-call radiologist is contacted and a CT head scan is arranged.

One hour later the patient extubates himself without warning. He is referred to medicine as his GCS is still 13/15. His CT demonstrates no acute intracranial pathology. By the time you arrive in the Emergency Department his GCS has improved to 15/15 and he is demanding to go home.

Which drug is most likely to be implicated?

- Diazepam
- Methoxetamine
- Gamma-hydroxybutyric acid (GHB)
- Heroin
- Methamphetamine

Submit answer

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Which drug is most likely to be implicated?

Diazepam	11%
Methoxetamine	7%
Gamma-hydroxybutyric acid (GHB)	51%
Heroin	21%
Methamphetamine	10%

GHB or 'Grievous Bodily Harm' is a colourless, odourless, bitter-tasting substance that acts as a CNS depressant. It is abused for its ability to induce euphoria, amnesia, and hypnosis. It is popular amongst party-goers and achieved notoriety for its use as a date-rape drug.

GHB toxicity occurs secondary to its effects as a CNS depressant. Patients typically present with coma, respiratory depression, mild bradycardia, and vomiting. The key feature of GHB toxicity is its short recovery time. Patients typically recover in 6 hours and toxicity is often maximal on presentation. Recovery often occurs spontaneously in the Emergency Department and patients have been known to self-extubate.

Heroin and diazepam could both produce a similar toxidrome. Recovery is generally more gradual, and one would expect naloxone to reverse a heroin overdose.

Methamphetamine is a stimulant. Clinical features include agitation, tachycardia, hypertension, mydriasis, and hyperthermia.

Methoxetamine is an analogue of the dissociative anaesthetics ketamine and phencyclidine (PCP). In addition to dissociative phenomena, it also causes tachycardia, hypertension, confusion, and mydriasis. Patients may also present with an acute cerebellar syndrome.

Discuss (6) | Improve

Next question >

Novel psychoactive substances ★

Novel psychoactive substances is the medical term for the many new substances which are chemically related to established recreational drugs such as MDMA and cannabis. They are often referred to as 'legal highs' although this is a misnomer in the UK, as their distribution and sale have been illegal since 2016.

The information below describes some of the common types:

Stimulants

- similar to MDMA, amphetamines and cocaine, resulting in increased levels of serotonin, dopamine and noradrenaline, resulting in a 'high' and feeling of euphoria
- a common example is a stimulant NPS is mephedrone ('bath salts','M-CAT'.'meow meow'). It is a cathinone and structurally similar to khat, a plant found in East Africa
- another example is benzylpiperazine ('Exodus', 'Legal X', 'Legal E')
- typically swallowed as a pill/powder ('bombing') or snorted
- adverse effect profile similar to MDMA/cocaine, with the risk of serotonin syndrome

Cannabinoids

- termed synthetic cannabinoid receptor agonists
- commonly referred to as 'spice'
- typically sprayed on to herbal mixtures which are then smoked. Also available in liquid form which is then inhaled using e-cigarettes
- similar adverse effects to cannabis

Hallucinogenics

- can be either dissociatives and psychedelics

- dissociatives produce a similar effect to ketamine, with a sense of not being connected to the physical body or time. A common dissociative NPS is methoxetamine ('mexxy')
- psychedelics have a similar effect to LSD although NPS versions may also be a stimulant

Depressant

- can be either opioid or benzodiazepine-based
- usually taken as a pill or a powder
- often structurally very similar to the original drug class, hence the adverse effects are similar
- benzodiazepine NPS often have a significantly longer half-life

Other substances include:

- Gamma-hydroxybutyric acid (GHB) and gamma-butyrolactone (GBL): 'G', 'Geebs' or 'Liquid Ecstasy'
- GHB causes respiratory depression. When taken with other respiratory depressants, most commonly alcohol, this can be potentially life threatening
- Nitrous oxide: 'Hippie crack'

For a more detailed overview please see the excellent review in BMJ 2017;356:i6848

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

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A 35-year-old man is brought to the emergency department. He was a bystander at a house fire, who called the fire and ambulance services. The female resident of the property had escaped the fire but was found collapsed in the front garden. The bystander started cardio-pulmonary resuscitation including mouth-to-mouth breathing until the ambulance crew arrived.

Whilst the emergency services were dealing with the fire and unconscious resident, the 35-year-old man starts to feel 'light-headed'. The ambulance crew bring him to the emergency department as a precaution.

He is quickly reviewed by the emergency team, who refer this gentleman because of ongoing symptoms and feel he needs a longer period of observation in the medical assessment unit. They handover there was no clinical features to suggest inhalation injury from the fire.

Upon your review, the patient has deteriorated. He is confused and unable to provide any history. You are able to perform a limited examination which reveals a temperature of 37.5ºC, heart rate 38 beats/min, blood pressure 80/60 mmHg, respiratory rate 10/min and oxygen saturation of 98% on room air. Examination of the chest reveals equal vesicular breath sounds, no added sounds and dual heart sounds with no murmurs. Neurological assessment reveals confusion, a reduced level of consciousness and dilated pupils bilaterally.

You take an urgent arterial blood gas and prescribe a bolus of crystalloid fluid. A 12 lead ECG shows sinus bradycardia. Whilst awaiting the results of the arterial blood gas, the patient develops generalised convulsions.

You and the emergency team work together to stabilise the patient - he is intubated, placed on mechanical ventilation and vasopressors are commenced to support his blood pressure. During this time, you are presented with the results of the arterial blood gas:

pH	7.11
PaO2	100mmHg
SaO2	96% on room air
PaCo2	28mmHg
Bicarbonate	7 mmol/L
Chloride	105 mmol/L
Lactate	8 mmol/L
Carboxyhaemoglobin	0.7%

Na+	138 mmol/l
K+	4.5 mmol/l

The full blood count and biochemistry results are still awaited.

Which of the following is the most appropriate management for this patient?

- ○ Urgent computed tomography (CT) imaging of the brain ×
- ○ Atropine boluses, followed by atropine infusion ×
- ○ Pralidoxime ×
- ○ Benztropine ×
- ○ Sodium thiosulfate ×

Submit answer

Reference ranges

Score: **15.1%**

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Lactate	8 mmol/L
Carboxyhaemoglobin	0.7%

Na+	138 mmol/l
K+	4.5 mmol/l

The full blood count and biochemistry results are still awaited.

Which of the following is the most appropriate management for this patient?

Urgent computed tomography (CT) imaging of the brain	4%
Atropine boluses, followed by atropine infusion	23%
Pralidoxime	14%
Benztropine	6%
Sodium thiosulfate	**53%**

Whist carbon monoxide poisoning is most associated with exposure to house/industrial fires, cyanide toxicity from burning of synthetic materials should also be considered

Important for me Less important

This man has cyanide poisoning. Performing mouth to mouth resuscitation has led to the oral transmission of cyanide molecules from the resident. The absorption of cyanide is slower via the oral route as compared to inhalation, which explains why this patient had a relatively 'delayed' presentation of cyanide poisoning. Also, exposure to small amounts of cyanide can lead to a longer duration of symptoms before cardiopulmonary arrest. The antidote for cyanide poisoning is intravenous sodium thiosulfate (+/- hydroxocobalamin).

Fire is the most common source of cyanide exposure in industrialised countries. Mouth-to-mouth resuscitation is contraindicated in cyanide poisoning because of risk to the provider of CPR.

The other differential diagnosis, in this case, is carbon monoxide inhalation. However, seizures are common in cyanide poisoning but are rare in carbon monoxide poisoning. Cyanide poisoning also causes mydriasis. whereas carbon monoxide does not affect the pupils. Administration of 100% oxygen is indicated for both carbon monoxide and cyanide poisoning.

In normal human physiology, detoxification of cyanide occurs via rhodanese, an enzyme found in many tissues but particularly in the liver and muscle. Rhodanese converts cyanide to thiocyanate, a water-soluble molecule excreted in the urine.

In poisoning, rhodanese is saturated and unable to remove the surplus cyanide molecules. Sodium

thiosulfate serves as a sulfur donor in the reaction catalyzed by rhodanese. Hence administration of sodium thiosulfate increases cyanide to thiocyanate conversion. Hydroxocobalamin (vitamin B12) has also been approved for the treatment of cyanide poisoning.

Although rare, you should think of cyanide poisoning in any patient with unexplained severe lactic acidosis, coma, bradycardia, and normal oxygenation.

Option 1 - treating the poisoning should take priority over investigating the reduced level of consciousness/seizures with brain imaging

Option 2 - this is part of the treatment algorithm for organophosphate poisoning

Option 3 - this is also part of the treatment algorithm for organophosphate poisoning

Option 4 - this is part of the treatment algorithm for dystonic reactions that may be secondary to anti-psychotic overdose

Option 5 - the correct answer

Discuss (5) Improve

Next question

Cyanide poisoning

Cyanide may be used in insecticides, photograph development and the production of certain metals. Cyanide inhibits the enzyme cytochrome c oxidase, resulting in cessation of the mitochondrial electron transfer chain.

Presentation

- 'classical' features: brick-red skin, smell of bitter almonds
- acute: hypoxia, hypotension, headache, confusion
- chronic: ataxia, peripheral neuropathy, dermatitis

Management

- supportive measures: 100% oxygen
- definitive: hydroxocobalamin (intravenously), also combination of amyl nitrite (inhaled), sodium nitrite (intravenously), and sodium thiosulfate (intravenously)

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Cyanide poisoning

Pixorize - YouTube 4 0

Cyanide poisoning

Kloss and Bruce - YouTube 1 1

Report broken media

Score: **18.1%**

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A 33-year-old woman with bipolar affective disorder attends as she is struggling to conceive. She also complains of severe fatigue, palpitations, and a worsening acneiform facial rash.

On review, you note that she has been taking olanzapine for over ten years.

Which of the following is she at increased risk due to long-term use of this medication?

- Acne
- Agranulocytosis
- Polycystic ovarian syndrome
- Prolonged QTc
- Type 2 diabetes mellitus

Submit answer

Reference ranges

Score: **15.1%**

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A 33-year-old woman with bipolar affective disorder attends as she is struggling to conceive. She also complains of severe fatigue, palpitations, and a worsening acneiform facial rash.

On review, you note that she has been taking olanzapine for over ten years.

Which of the following is she at increased risk due to long-term use of this medication?

Acne	1%
Agranulocytosis	18%
Polycystic ovarian syndrome	14%
Prolonged QTc	12%
Type 2 diabetes mellitus	56%

Long-term use of olanzapine is a significant risk factor for the development of type 2 diabetes mellitus

Important for me Less important

Type 2 diabetes mellitus is correct. Long-term olanzapine use is a significant risk factor for developing type 2 diabetes.

Acne is incorrect. There is no evidence to show a link between the development of acne and olanzapine use.

Polycystic ovarian syndrome is incorrect. There is no evidence to suggest an increased risk of polycystic ovarian syndrome as a result of olanzapine use. Traditional neuroleptics can lead to raised levels of prolactin which causes relative infertility. Olanzapine has only minimal effect on prolactin levels.

Agranulocytosis is incorrect. Some antipsychotic medications including clozapine, tetracyclic anti-depressants, and phenothiazines have been shown to cause agranulocytosis, but this is not the case with olanzapine. There are a small handful of case reports showing blood dyscrasias with olanzapine and so monitoring full blood counts is still recommended.

Prolonged QTc is not seen with olanzapine use but can be seen with other neuroleptic medications such as chlorpromazine, fluoxetine, haloperidol, and amitriptyline.

Discuss (1) Improve

Next question ›

Drug-induced impaired glucose tolerance

Drugs which are known to cause impaired glucose tolerance include:

- thiazides, furosemide (less common)
- steroids
- tacrolimus, ciclosporin
- interferon-alpha
- nicotinic acid
- antipsychotics

Beta-blockers cause a slight impairment of glucose tolerance. They should also be used with caution in diabetics as they can interfere with the metabolic and autonomic responses to hypoglycaemia

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

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A 45 woman is admitted with fever, night sweats and weight loss. She has a past medical history of type 1 diabetes. At a recent retinal screen there were microaneurysms and dot and blot haemorrhages noted on the periphery of both her discs. She has never noted any visual problems.

A sputum sample is positive for acid fast bacilli and she is subsequently started on Rifampicin, Isoniazid, Pyrazinamide, ethambutol and pyridoxine.

On the same admission she is diagnosed with HIV. Her CD4 count is 140 cells/μL. She is started cotrimoxazole and a few weeks later starts antiretroviral therapy with Efavirenz, emtricitabine and tenofovir.

She makes a good recovery and is discharged from hospital. A few months later she complains of increasingly blurry vision. Fundoscopy shows pale optic discs, microaneurysms and blot haemorrhages.

What is the most likely cause for her visual symptoms?

- CMV retinitis
- Ethambutol
- Efavirenz
- Diabetic retinopathy
- Pyrizinamide

Submit answer

Reference ranges

Score: **15.1%**

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She makes a good recovery and is discharged from hospital. A few months later she complains of increasingly blurry vision. Fundoscopy shows pale optic discs, microaneurysms and blot haemorrhages.

What is the most likely cause for her visual symptoms?

CMV retinitis	13%
Ethambutol	72%
Efavirenz	4%
Diabetic retinopathy	9%
Pyrizinamide	2%

Ethambutol is the correct answer here and is well known to cause optic neuritis. Diabetic retinopathy is unlikely to cause such an acute deterioration considering her recent retinal screen. CMV retinitis is unlikely to cause this picture and efavirenz and pyrazinamide do not have ocular side effects.

Tuberculosis: drug side-effects and mechanism of action

Rifampicin

- mechanism of action: inhibits bacterial DNA dependent RNA polymerase preventing transcription of DNA into mRNA
- potent liver enzyme inducer
- hepatitis, orange secretions
- flu-like symptoms

Isoniazid

- mechanism of action: inhibits mycolic acid synthesis
- peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
- hepatitis, agranulocytosis
- liver enzyme inhibitor

Pyrazinamide

- mechanism of action: converted by pyrazinamidase into pyrazinoic acid which in turn inhibits fatty acid synthase (FAS) I
- hyperuricaemia causing gout
- arthralgia, myalgia
- hepatitis

Ethambutol

- mechanism of action: inhibits the enzyme arabinosyl transferase which polymerizes arabinose into arabinan
- optic neuritis: check visual acuity before and during treatment
- dose needs adjusting in patients with renal impairment

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

0 0

2016 Tuberculosis guidelines

Suggest link Report broken link

Media

Tuberculosis

Osmosis - YouTube 0 0

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A 46-year-old male was transferred to the Intensive Care Unit (ICU) as an emergency admission. He had been scheduled to undergo an elective open cholecystectomy under general anaesthetic. He had been intubated and ventilated successfully, but a few minutes into the operation he had become tachycardic, with his heart rate rising from a resting rate of 72 bpm to a rate of 142bpm. His oxygen saturations dropped down to 92% on 15 litres of oxygen per minute, and his blood pressure rose from his baseline of 114/78 mmHg up to 162/98 mmHg. In addition, his end tidal CO2 concentration rose, despite several checks to ensure that the tracheal tube was correctly sited. His past medical history was unremarkable except for an appendicectomy under general anaesthetic aged 26 yrs old.

The operation was abandoned. Upon his arrival to ICU, he was intubated and ventilated, maintaining an oxygen saturation of 96% on 15 litres per minute of oxygen. He appeared flushed and had significant muscle rigidity. His blood pressure had risen to 174/102 mmHg, and his heart rate was 136bpm. His temperature was 38.2 C. Examination of the cardiovascular revealed vasodilated peripheries with a bounding pulse and normal heart sounds. Auscultation of the lungs revealed good air entry in all zones and a correctly cited tracheal tube. Examination of the abdomen was unremarkable. Examination of the neurological system confirmed the presence of equal and reactive pupils, with generalised muscle hypertonicity and masseter muscle spasm. A central venous catheter and arterial line were inserted. The central venous pressure was 9 cm.

An arterial blood gas sample on 15 litres of oxygen was taken:

pH	7.26
HCO3	18 mmol/l
Pa02	17 kPa
PaC02	7.6 kPa
Na+	139 mmol/l
K+	7.1 mmol/l

What is the next best immediate management step?

- Commence IV labetalol infusion
- Commence IV meropenem

- [ ] Commence high dose IV hydrocortisone
- [ ] Commence IV dantrolene
- [ ] Arrange immediate therapeutic hypothermia

Submit answer

Reference ranges

Score: **15.1%**

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An arterial blood gas sample on 15 litres of oxygen was taken:

pH	7.26
HCO3	18 mmol/l
Pa02	17 kPa
PaC02	7.6 kPa
Na+	139 mmol/l
K+	7.1 mmol/l

What is the next best immediate management step?

- Commence IV labetalol infusion 1%
- Commence IV meropenem 1%

Commence high dose IV hydrocortisone	4%
Commence IV dantrolene	89%
Arrange immediate therapeutic hypothermia	5%

This patient has developed malignant hyperthermia as a result of his anaesthetic. In addition to stopping the responsible anaesthetic agent, the best immediate management step involves commencing IV dantrolene. Early commencement of dantrolene has greatly reduced mortality associated with malignant hyperthermia and should be administered as soon as the diagnosis is suspected. Note that hyperthermia is a relatively late feature of malignant hyperthermia, and earlier features include hypoxia, hypercarbia, masseter muscle spasm and hypertonicity.

Discuss (6) | Improve

Next question

Malignant hyperthermia

Overview

- condition often seen following administration of anaesthetic agents
- characterised by hyperpyrexia and muscle rigidity
- cause by excessive release of Ca2+ from the sarcoplasmic reticulum of skeletal muscle
- associated with defects in a gene on chromosome 19 encoding the ryanodine receptor, which controls Ca2+ release from the sarcoplasmic reticulum
- susceptibility to malignant hyperthermia is inherited in an autosomal dominant fashion
- neuroleptic malignant syndrome may have a similar aetiology

Causative agents

- halothane
- suxamethonium
- other drugs: antipsychotics (neuroleptic malignant syndrome)

Investigations

- CK raised
- contracture tests with halothane and caffeine

Management

- dantrolene - prevents Ca2+ release from the sarcoplasmic reticulum

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 58-year-old man presents with several months of feeling generally tired, cold and experiencing some weight gain. His past medical history includes atrial fibrillation, for which he has taken amiodarone for several years.

Blood tests are performed, which show the following:

Thyroid-stimulating hormone (TSH)	9.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	6.7 pmol/L	(9.0 - 18)

Given the above, what is most appropriate?

- Continue amiodarone and start carbimazole
- Continue amiodarone and start levothyroxine
- Stop amiodarone and monitor
- Stop amiodarone and start levothyroxine
- Stop amiodarone and start prednisolone

Submit answer

Reference ranges

Score: **15.1%**

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A 58-year-old man presents with several months of feeling generally tired, cold and experiencing some weight gain. His past medical history includes atrial fibrillation, for which he has taken amiodarone for several years.

Blood tests are performed, which show the following:

Thyroid-stimulating hormone (TSH)	9.2 mU/L	(0.5-5.5)
Free thyroxine (T4)	6.7 pmol/L	(9.0 - 18)

Given the above, what is most appropriate?

Continue amiodarone and start carbimazole	2%
Continue amiodarone and start levothyroxine	60%
Stop amiodarone and monitor	12%
Stop amiodarone and start levothyroxine	10%
Stop amiodarone and start prednisolone	16%

In amiodarone induced hypothyroidism, amiodarone can be continued with levothyroxine

Important for me Less important

The scenario above demonstrates an example of amiodarone-induced hypothyroidism. This is common, with around 1 in 6 patients taking amiodarone developing some form of thyroid dysfunction. As well as **treating the deficit with levothyroxine, amiodarone can be continued** in these patients, and so this is the correct answer.

Continuing amiodarone and starting carbimazole is inappropriate. Carbimazole would be appropriate for thyrotoxicosis, not hypothyroidism. In thyrotoxicosis, amiodarone should be stopped, unlike hypothyroidism, and so continuing would be incorrect. If type 1 - excess iodine-induced thyroid hormone synthesis and a goitre present, carbimazole is an appropriate option.

Stopping amiodarone and monitoring is not the preferred management option here. Amiodarone is important with regards to controlling this patient's atrial fibrillation. Also,

levothyroxine should be administered to treat hypothyroidism.

Stopping amiodarone and starting levothyroxine is not quite right. Although commencing levothyroxine is appropriate, amiodarone should be continued.

Stopping amiodarone and starting prednisolone is incorrect. This would be suitable for type 2 thyrotoxicosis. Type 2 is defined as amiodarone-related destructive thyroiditis with an absent goitre.

👍 👎 💬 Discuss (3) Improve

Next question ›

Amiodarone and the thyroid gland ★

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link

Report broken link

Score: **18.1%**

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A 48-year-old man is referred to the outpatient department by his GP, having experienced tremor, heat intolerance and 2 kg weight loss over the last 6 weeks. His past medical history includes having atrial fibrillation for which he takes warfarin and amiodarone. He is a non-smoker and drinks on average 10 units of alcohol per week.

Blood tests are performed and reveal:

Hb	142 g/l	
Platelets	220 * 10^9/l	
WBC	7.2 * 10^9/l	
Na^+	140 mmol/l	
K^+	4.2 mmol/l	
Urea	4.5 mmol/l	
Creatinine	45 µmol/l	
Thyroid stimulating hormone (TSH)	0.03 mu/l	
Free thyroxine (T4)	29 pmol/l	
Total T3 (TT3)	252 ng/dL	Normal range 75 -200 ng/dL

A colour flow Doppler sonography of the thyroid is performed and shows absent vascularity and gland destruction.

What is the most likely diagnosis?

- Type 1 amiodarone-induced thyrotoxicosis
- Type 2 amiodarone-induced thyrotoxicosis
- Grave's disease
- Hashimotos thyroiditis
- De Quervains thyroiditis

Submit answer

Reference ranges

Score: **15.1%**

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Total T3 (TT3)	252 ng/dL	Normal range 75 -200 ng/dL

A colour flow Doppler sonography of the thyroid is performed and shows absent vascularity and gland destruction.

What is the most likely diagnosis?

Type 1 amiodarone-induced thyrotoxicosis	19%
Type 2 amiodarone-induced thyrotoxicosis	78%
Grave's disease	1%
Hashimotos thyroiditis	1%
De Quervains thyroiditis	1%

Type 2 amiodarone-induced thyrotoxicosis typically occurs in patients without underlying thyroid

disease and is the result of the toxic effect of amiodarone on the thyroid follicular cells, causing a destructive thyroiditis that results in excess release of preformed T4 and T3 into the circulation. The condition has two phases a thyrotoxic phase which can last weeks to months, which is often followed by a hypothyroid phase and an eventual recovery in most patients.

Type 1 amiodarone-induced thyrotoxicosis normally occurs in patients with underlying thyroid pathology like Graves disease. In these patients, there is accelerated thyroid hormone synthesis secondary to the iodide load from the amiodarone therapy.

👍 👎 💬 Discuss (3) Improve

Next question ›

Amiodarone and the thyroid gland ★

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

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Next question

Textbooks

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5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link

Report broken link

Score: **18.1%**

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A 25-year-old woman presents to the Emergency department some 90 mins after taking 50x300mg aspirin tablets. She feels nauseated, has vomited once, complains of severe tinnitus, and is hyperventilating. She has no past medical history of note and takes no regular medications. Examination reveals a blood pressure of 105/70 mmHg, pulse is 87 beats per minute and regular. Chest is clear, respiratory rate is elevated at 35/min. She has been given activated charcoal on admission. 1 litre of normal saline given intravenously over 1hr is in progress.

investigations

Hb	125 g/l
Platelets	189 * 10^9/l
WBC	10.2 * 10^9/l

Na^+	145 mmol/l
K^+	3.1 mmol/l
Urea	6.1 mmol/l
Creatinine	90 µmol/l
pH	7.54
Serum salicylate	4.2 mmol/l

Which of the following is the most appropriate next step?

- IV furosemide
- IV sodium bicarbonate
- Oral diazepam
- Referral for haemodialysis
- Repeated activated charcoal

Submit answer

Reference ranges

Score: **15.1%**

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Urea	6.1 mmol/l
Creatinine	90 µmol/l
pH	7.54
Serum salicylate	4.2 mmol/l

Which of the following is the most appropriate next step?

IV furosemide	1%
IV sodium bicarbonate	54%
Oral diazepam	2%
Referral for haemodialysis	33%
Repeated activated charcoal	9%

Administration of sodium bicarbonate is recommended to drive alkalinisation of urine, even where there is respiratory alkalosis in aspirin overdose. It is therefore the correct next step here.The

recommended dose If the salicylate concentration in an adult is above 3.6 mmol/L is 225 mmol sodium bicarbonate, (225 mL of 8.4% over 60 minutes or 1.5 L of 1.26% over two hours).

Forced diuresis with furosemide is not recommended as large fluid shifts may increase the risk of pulmonary oedema associated with aspirin overdose. Oral diazepam is not appropriate and would potentially just shorten the time to development of acidosis. Haemodialysis is considered when salicylate concentration is greater than 5.1 mmol/l.

👍 👎 💬 Discuss (4) Improve

Next question ›

Salicylate overdose ★

A key concept for the exam is to understand that salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis. In children metabolic acidosis tends to predominate.

Features

- hyperventilation (centrally stimulates respiration)
- tinnitus
- lethargy
- sweating, pyrexia*
- nausea/vomiting
- hyperglycaemia and hypoglycaemia
- seizures
- coma

Treatment

- general (ABC, charcoal)
- urinary alkalinization with intravenous sodium bicarbonate - enhances elimination of aspirin in the urine
- haemodialysis

Indications for haemodialysis in salicylate overdose

- serum concentration > 700mg/L
- metabolic acidosis resistant to treatment
- acute renal failure
- pulmonary oedema

- seizures
- coma

*salicylates cause the uncoupling of oxidative phosphorylation leading to decreased adenosine triphosphate production, increased oxygen consumption and increased carbon dioxide and heat production

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medscape 2 0

Salicylate Toxicity review

Life in The Fast Lane 1 0

Salicylate toxicity

Suggest link Report broken link

Score: **18.1%**

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A 52-year-old gentleman presents after hearing a popping sound followed by acute onset right ankle pain. On examination he is unable to plantar flex his right ankle. He denies any recent strenuous exercise. He has never had any joint problems but does tell you he has recently been treated for a urinary tract infection. Which one of these antibiotics is likely have contributed to his presentation?

- Erythromycin
- Trimethoprim
- Nitrofurantoin
- Ciprofloxacin
- Co-amoxiclav

Submit answer

Reference ranges

Score: **15.1%**

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Erythromycin	3%
Trimethoprim	2%
Nitrofurantoin	5%
Ciprofloxacin	90%
Co-amoxiclav	1%

Ciprofloxacin may lead to tendinopathy

Important for me Less important

A rare side effect of ciprofloxacin is tendon inflammation +/- tendon rupture as listed in the BNF. With a history suggestive of tendon rupture remember to ask about recent antibiotic usage.

https://www.medicinescomplete.com/mc/bnf/current/PHP3665-ciprofloxacin.htm#PHP56148-sideEffects-topic

Discuss (1) Improve

Next question

Quinolones

Quinolones are a group of antibiotics which work by inhibiting DNA synthesis and are bactericidal in nature. Examples include:

- ciprofloxacin

- levofloxacin

Mechanism of action
- inhibit topoisomerase II (DNA gyrase) and topoisomerase IV

Mechanism of resistance
- mutations to DNA gyrase, efflux pumps which reduce intracellular quinolone concentration

Adverse effects
- lower seizure threshold in patients with epilepsy
- tendon damage (including rupture) - the risk is increased in patients also taking steroids
- cartilage damage has been demonstrated in animal models and for this reason quinolones are generally avoided (but not necessarily contraindicated) in children
- lengthens QT interval

Contraindications
- Quinolones should generally be avoided in women who are pregnant or breastfeeding
- avoid in G6PD

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

10 2

Quinolones

Suggest link Report broken link

Score: **18.1%**

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A 37-year-old man attends endocrinology clinic for assessment of the unwanted effects of long-term anabolic steroid use. The patient had been a competitive bodybuilder, who had used anabolic steroids to enhance his training regime, but now stated that he had 'retired' from bodybuilding last week and so stopped using steroids. The patient was concerned about the long-term health consequences of his previous use.

The patient described a 10-year period of anabolic steroid use. Typically he would take a daily oral formulation of a synthetic testosterone supplemented by an intramuscular injection of a longer acting agent every few weeks. The patient would take breaks from anabolic steroid use intermittently throughout the year, to reduce unwanted effects and also to evade anti-doping testing arranged by the organisers of the competitions in which he competed. The patient stated that he had never shared or reused needles when injecting himself with anabolic steroids.

The patient reported a range of unwanted effects he had developed secondary to his anabolic steroid use. These included severe acne affecting the patient's face and chest, intermittent symptoms of gastrointestinal dysfunction, and male pattern baldness. When asked directly, the patient reported that he had suffered from erectile dysfunction and scrotal discomfort towards the end of his use of anabolic steroids. The patient did not report any other concerns, in particular, he denied symptoms associated with heart or liver disease. The patient had no other significant past medical history and disclosed being a former user of recreational drugs, including cocaine.

Examination of the patient's cardiovascular and respiratory symptoms was unremarkable except for moderate pitting oedema to the level of the mid-tibia bilaterally. Examination of the abdomen noted mild bilateral gynaecomastia but no other signs of chronic liver disease. The patient's testicular volume was estimated as 18 ml bilaterally. A brief mental state examination did not reveal any evidence of a significant mood or anxiety disorder.

HbA1C	47 mmol / mol (reference < 42)
Fasting LDL cholesterol	4.5 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.0 mmol / L (reference > 1.2)
Prolactin	501 mU / L (reference 80 - 400)
Luteinising hormone	1.4 IU / L (reference 1.8 - 8.6)
Follicle-stimulating hormone	1.2 mU / ml (reference 1.5 - 12.4)
Transthoracic echocardiogram	Normal systolic and diastolic function; no left ventricular hypertrophy; normal valvular function

Which of the patient's unwanted effects will be irreversible with cessation of steroid use?

- ○ Pitting oedema ×
- ○ Erectile dysfunction ×
- ○ Scrotal pain ×
- ○ Male pattern baldness ×
- ○ Gastrointestinal dysfunction ×

Submit answer

Reference ranges

Score: **15.1%**

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Transthoracic echocardiogram	Normal systolic and diastolic function; no left ventricular hypertrophy; normal valvular function

Which of the patient's unwanted effects will be irreversible with cessation of steroid use?

Anabolic steroid use is associated with a wide variety of unwanted effects. Morbidity and mortality from cardiac diseases are increased, although the precise mechanism of this effect is unknown. In addition, serious liver disease - for example, hepatocellular carcinoma - is also associated with anabolic steroid use. There are a variety of less dangerous but troubling unwanted effects as described below, some of which will be irreversible with cessation of anabolic steroid use. Due to the potential risk to long-term health, anabolic steroid users should be strongly advised to stop their use. No tapering of the dose is required. Active or former users should be monitored lifelong for evidence of complications.

Reversible unwanted effects of anabolic steroids include:

- Increased appetite
- Gastrointestinal dysfunction
- Mood swings
- Anxiety
- Acne
- Oedema
- Libido change
- Scrotal pain
- Erectile dysfunction
- Menstrual irregularities

Irreversible unwanted effects of anabolic steroids include:

- Hirsutism
- Voice pitch changes
- Male pattern baldness
- Skin striae or keloid scarring
- Chest pain
- Clitoral hypertrophy
- Short stature due to premature fusion of growth plates

It is unclear whether other unwanted effects are reversible with cessation of anabolic steroids; for example, gynaecomastia, testicular atrophy and infertility. Some limited studies suggest that normal sperm production and fertility return following cessation of anabolic steroid use, although on a timescale ranging from 4 months to 5 years.

The patient also has a typical pattern of hormonal irregularities associated with anabolic steroid use. A deranged lipid profile and impaired glucose tolerance are also frequently observed. The patient's echocardiogram is reassuringly normal, which suggests that his leg oedema will resolve with cessation of anabolic steroid use.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

Discuss (5) Improve

Next question ›

Anabolic steroid use

Anabolic steroid use is associated with several serious long-term health consequences. Cardiac morbidity and mortality are increased by anabolic steroid use, although the precise mechanism of this effect is unclear. Hepatic side effects also occur secondary to chronic vascular injury: these include hepatocellular carcinoma and hepatic adenoma. Psychiatric illness is also commonly co-morbid with anabolic steroid use. Additionally, users who inject anabolic steroids have an increased risk of blood-borne viruses if needles are shared between individuals.

Due to the above concerns, patients should be strongly counselled to stop using anabolic steroids. There is no requirement for tapering of doses. Many of the above blood test abnormalities normalise once anabolic steroid consumption ceases. The expert recommendation is for lifelong monitoring for potential complications, initially annually with frequency reducing once blood markers normalise and in the absence of apparent adverse effects.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

AI + Q 123

Next question ›

B I A T

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 23-year-old male is brought to the ED via ambulance after a seizure. His friends reported an episode of jerking of all four limbs lasting 1-2 minutes associated with urinary incontinence.

He has vomited twice in the ED and is complaining of muscle aches.

On examination the patient is agitated, confused and appears to be hallucinating. Myoclonic jerks are present. The chest is clear to auscultation and oxygen saturations are 98% on room air. Heart sounds are normal, the pulse rate is 130bpm and the blood pressure is 161/84mmHg.

An ECG reveals sinus tachycardia. The pupils measure 8mm and are equal and reactive. The patient's temperature is 36.6ºC.

Blood tests reveal:

Hb	138 g/l
Platelets	362 * 10^9/l
WBC	11.2 * 10^9/l
Na^+	135 mmol/l
K^+	2.7 mmol/l
Urea	12.6 mmol/l
Creatinine	187 µmol/l
Bilirubin	18 µmol/l
ALP	98 u/l
ALT	53 u/l
γGT	27 u/l
Albumin	32 g/l

What is the most likely cause for this patient's presentation?

- Toluene solvent toxicity
- Amyl nitrate toxicity

- Gamma hydroxybutyric acid toxicity
- Synthetic cannabinoid toxicity
- Heroin toxicity

Submit answer

Reference ranges

Score: **15.1%**

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γGT	27 u/l
Albumin	32 g/l

What is the most likely cause for this patient's presentation?

Toluene solvent toxicity	12%
Amyl nitrate toxicity	16%

Gamma hydroxybutyric acid toxicity	27%
Synthetic cannabinoid toxicity	34%
Heroin toxicity	12%

The hallucinations, seizure, myoclonus, mydriasis, hypertension, acute kidney injury and hypokalaemia are consistent with synthetic cannabinoid toxicity.

Features of toluene toxicity can be similar however irritation to the eyes, nose and respiratory tract is often seen from inhalation in toluene abuse.

Amyl nitrate is a vasodilator so toxicity tends to result in hypotension rather than hypertension as in this case. Other features of amyl nitrate toxicity include blurred vision, xanthopsia and haemoptysis.

Features of gamma hydroxybutyric acid (GHB) toxicity include CNS and respiratory depression, hypersalivation, bradycardia and hypotension.

Heroin toxicity typically results in depression of the respiratory and central nervous systems and pin-point pupils (rather than mydriasis in this case).

(www.toxbase,org)

Discuss (5) | Improve

Next question >

Cannabis ★

Features of synthetic cannabinoid toxicity include:

CNS: agitation, tremor, anxiety, confusion, somnolence, syncope, hallucinations, changes in perception, acute psychosis, nystagmus, convulsions and coma.

Cardiac: tachycardia, hypertension, chest pain, palpitations, ECG changes.

Renal: acute kidney injury.

Muscular: hypertonia, myoclonus, muscle jerking and myalgia.

Other: cold extremities, dry mouth, dyspnoea, mydriasis, vomiting and hypokalaemia

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Cannabis Dependence

Osmosis - YouTube 2 0

Report broken media

Score: **18.1%**

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A 69-year-old man with a history of chronic obstructive pulmonary disease is admitted to the Emergency department with an exacerbation. His GP has started ciprofloxacin some 3 days earlier. Other medication of note includes high dose salmeterol fluticasone, oral theophylline and tiotropium. Over the past 24hrs he has begun to vomit and feels light-headed. Of note is that his blood pressure is 132/88 mmHg with a postural drop of 15 mmHg, and his serum potassium is 3.0 mmol/l.

Which of the following is the most appropriate intervention?

- Stop salmeterol fluticasone
- Stop theophylline
- Stop tiotropium
- Stop ciprofloxacin
- Start oral potassium replacement

Submit answer

Reference ranges

Score: **15.1%**

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Which of the following is the most appropriate intervention?

Stop salmeterol fluticasone	6%
Stop theophylline	53%
Stop tiotropium	1%
Stop ciprofloxacin	35%
Start oral potassium replacement	5%

Stop theophylline when CYP450 inhibitors are prescribed

Important for me Less important

Theophylline is metabolised by the CYP1A2 isoenzyme. Ciprofloxacin is a moderate inhibitor of CYP1A2, which can drive accumulation of theophylline. Symptoms of toxicity include nausea, vomiting, hyperventilation and marked hypokalaemia, as is seen here. The most important intervention is withholding of the theophylline.

Metabolism of salmeterol and tiotropium is not affected by co-administration of ciprofloxacin as such there is no reason for discontinuing these medications. Starting oral potassium replacement won't affect the build up of theophylline and won't therefore prevent further symptoms of theophylline toxicity. Clozapine, olanzapine, ropinirole and duloxetine are other examples of drugs metabolised by CYP1A2, accumulation of which may be driven by ciprofloxacin.

https://www.medicines.org.uk/emc/medicine/25756#INTERACTIONS

P450 drug interactions: more detail ★

Whilst you are expected to know in broad terms what are the main inhibitors and inducers of the P450 system it is unlikely that you will be asked detailed questions about the individual enzyme systems.

It is worthwhile noting that the most important and common reason for drug interactions is the P450 **CYP3A4 system**.

The table below shows the main enzyme systems that are affected by common drugs. There is clearly a lot of overlap within the various P450 enzymes.

P450 system	Substrates	Inhibitors	Inducers
CYP3A4	Macrolides Antiretrovirals Calcium channel blockers	Macrolides Protease inhibitors (including ritonavir) Imidazoles	Carbamazepine Phenytoin Phenobarbitone Rifampicin St John's Wort
CYP2D6	Tricyclic antidepressants Antipsychotics	SSRIs Ritonavir	
CYP2C9	Warfarin Sulfonylureas	Imidazoles Amiodarone Sodium valproate	Rifampicin
CYP1A2	Theophylline	Ciprofloxacin	Smoking Omeprazole
CYP2E1	Alcohol		Chronic alcohol Isoniazid

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 20-year-old female presents having taken 20 aspirin 500mg tablets in a suicide attempt. She has felt well since and is asymptomatic, and now regrets these actions. She has a background of borderline personality disorder for which she takes olanzapine and has never attempted suicide in the past. She was seen by her GP with a presumed respiratory tract infection one week ago and prescribed amoxicillin. Cardiovascular, respiratory and abdominal examinations are all unremarkable. Her blood gas reveals a ph 7.41, lactate 0.5mmol/L and laboratory bloods are unremarkable other than a salicylate level 4 hours post ingestion of 660mg/L.

Which management step should be taken next?

- Intravenous (IV) Sodium Chloride 0.9% ×
- Reassure and discharge ×
- IV Sodium bicarbonate 1.26% ×
- N-acetylcysteine infusion ×
- Dialysis ×

Submit answer

Reference ranges

Score: **15.1%**

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Which management step should be taken next?

Intravenous (IV) Sodium Chloride 0.9%	16%
Reassure and discharge	8%
IV Sodium bicarbonate 1.26%	53%
N-acetylcysteine infusion	10%
Dialysis	12%

Although this young lady demonstrates no obvious compromise from her overdose, guidelines suggest that salicylate levels greater than 450mg/L in adults represents moderate poisoning. Sodium bicarbonate used here alkalinises the urine which is more effective than a massive diuresis. Urine pH should be greater than 7.5, ideally 8.0-8.5.

Full guidelines can be found here.

Discuss (6) | Improve

Next question

Salicylate overdose

A key concept for the exam is to understand that salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis. In children metabolic acidosis tends to predominate.

Features

- hyperventilation (centrally stimulates respiration)
- tinnitus
- lethargy
- sweating, pyrexia*
- nausea/vomiting
- hyperglycaemia and hypoglycaemia
- seizures
- coma

Treatment

- general (ABC, charcoal)
- urinary alkalinization with intravenous sodium bicarbonate - enhances elimination of aspirin in the urine
- haemodialysis

Indications for haemodialysis in salicylate overdose

- serum concentration > 700mg/L
- metabolic acidosis resistant to treatment
- acute renal failure
- pulmonary oedema
- seizures
- coma

*salicylates cause the uncoupling of oxidative phosphorylation leading to decreased adenosine triphosphate production, increased oxygen consumption and increased carbon dioxide and heat production

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Medscape

2 0

Salicylate Toxicity review

Life in The Fast Lane

1 0

Salicylate toxicity

Suggest link

Report broken link

Score: **18.1%**

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A 77-year-old woman is admitted to the acute medical ward with a 3-day history of right loin pain and dysuria. A diagnosis of pyelonephritis is made and she is treated with intravenous gentamicin and ceftazidime.

Gentamicin levels are taken 8 hours post-dose:

Trough level	3.1 mg/L	(1 - 2)

What is the most appropriate next step in the management of this patient?

- Continue with dose and prescribe IV 0.9% sodium chloride
- Delay next dose of gentamicin
- Reduce dose of gentamicin
- Stop gentamicin
- Switch antibiotic to ciprofloxacin

Submit answer

Reference ranges

Score: **15.1%**

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A 77-year-old woman is admitted to the acute medical ward with a 3-day history of right loin pain and dysuria. A diagnosis of pyelonephritis is made and she is treated with intravenous gentamicin and ceftazidime.

Gentamicin levels are taken 8 hours post-dose:

Trough level	3.1 mg/L	(1 - 2)

What is the most appropriate next step in the management of this patient?

- Continue with dose and prescribe IV 0.9% sodium chloride — 4%
- **Delay next dose of gentamicin** — 56%
- Reduce dose of gentamicin — 36%
- Stop gentamicin — 3%
- Switch antibiotic to ciprofloxacin — 1%

For multiple daily dose regimens of gentamicin, if the pre-dose ('trough') concentration is high, the interval between doses must be increased. If the post-dose ('peak') concentration is high, the dose must be decreased

Important for me Less important

Patients receiving gentamicin therapy should have both pre-dose (trough) and post-dose (peak) levels checked. The recommended trough levels should be 1-2 mg/L, whilst the peak levels should be between 5-10 mg/L. A raised trough level means that there should be a **delay in the next dose of gentamicin**.

Continuing with the dose and prescribing IV 0.9% sodium chloride is inappropriate as this means that the patient could be receiving a harmful dose that could cause toxicity such as renal or otic impairment.

In contrast to raised trough levels, a raised peak level means that there should be a **reduced dose of gentamicin**.

There is no need to **stop gentamicin** at this stage. Instead, a raised trough level indicates an increased drug dosage interval as per the BNF. If the drug were to no longer be indicated or was evidenced to be causing significant harm or impairment in renal function, then careful discussion regarding its continuation would be needed.

Switch antibiotic to ciprofloxacin is incorrect. Rather than changing antibiotics, a raised trough level indicates that an increased dosing interval before the next administration of gentamicin is needed. It would be inappropriate to change antibiotics as there are risks for antimicrobial resistance.

Discuss (4) | Improve

Next question >

Gentamicin ★

Gentamicin is a type of aminoglycoside antibiotic. It is poorly lipid-soluble and is therefore given parentally (e.g. for infective endocarditis) or topically (e.g. for otitis externa).

Adverse effects

- ototoxicity
  - due to auditory or vestibular nerve damage
  - irreversible
- nephrotoxicity
  - accumulates in renal failure
  - gentamicin is preferentially taken up by the proximal renal tubular cells. Once inside these cells, it can accumulate to toxic levels
  - the toxicity is secondary to acute tubular necrosis
  - concomitant use of furosemide increases the risk
  - lower doses and more frequent monitoring is required

Contraindications

- myasthenia gravis

Dosing

- due to the significant ototoxic and nephrotoxic potential of gentamicin it is important to monitor plasma concentrations
- both peak (1 hour after administration) and trough levels (just before the next dose) are measured

- if the trough (pre-dose) level is high the interval between the doses should be increased
- if the peak (post-dose) level is high the dose should be decreased

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 19 year old girl is brought to the Emergency Department by a friend. She is a first year university student and her friends are concerned her behaviour has altered in the past few weeks. On assessment she is confused with an AMTS of 6/10. She has a flat affect. Cardiorespiratory examination is normal although the respiratory rate is 22 at rest. Oxygen saturations are 97% on air. Heart rate is 85bpm regular and blood pressure is 100/55 mmHg.

Examination of her neurological system reveals prominent paraesthesia in both feet with absent vibration sense and proprioception. The ankle and knee reflexes are absent and plantar responses are extensor. There is mild distal weakness in the legs with MRC grading 4/5. Examination of the cranial nerves shows no evidence of nystagmus but visual acuity is reduced to 6/20 bilaterally. She has an ataxic gait.

Her medical history is significant only for coeliac disease and she is taking no regular medications.

Routine blood results show:

Haemoglobin	7.6g/L	Sodium	137mmol/L	Magnesium	0.81mmol/L
Cell volume	103.2fL	Potassium	4.6mmol/L	CRP	4.5mg/L
White cells	$5.9x10^9$/L	Urea	1.9mmol/L	Thyroid tests	Normal
Neutrophils	$4.0x10^9$/L	Creatinine	55mol/L	HIV	Negative
Lymphocytes	$1.9x10^9$/L	Calcium (corr)	2.43mmol/L	Syphilis	Negative
Platelets	$300x10^9$/L	Phosphate	1.01mmol/L		

The patient's friend tells you she has recently started using recreational party drugs.

Which of the following inhaled recreational drugs most explains the above picture?

- Crack (cocaine)
- H (heroin)
- Hippie crack (nitrous oxide)
- Poppers (amyl nitrate)

Spice (synthetic cannabinoid)

Submit answer

Reference ranges

Score: **15.1%**

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The patient's friend tells you she has recently started using recreational party drugs.

Which of the following inhaled recreational drugs most explains the above picture?

Crack (cocaine)	3%
H (heroin)	3%
Hippie crack (nitrous oxide)	53%
Poppers (amyl nitrate)	26%

Spice (synthetic cannabinoid) 15%

Nitrous oxide use can precipitate severe vitamin B12 deficiency with pronounced neurological and haematological signs, particularly in susceptible individuals

Important for me Less important

The above vignette describes a state of profound vitamin B12 deficiency with clinical signs of subacute combined degeneration of the cord. The constellation of signs of ataxia, loss of deep tendon reflexes with dorsal column signs (loss of proprioception and vibration sense) and paraesthesias with positive Babinski response are known as myelosis funicularis and is highly suggestive of B12 deficiency. In this patient there are also signs of confusion and of reduced visual acuity which increase likelihood of B12 deficiency.

Nitrous oxide, also known as nitrous, hippie crack, laughing gas or whippet, is a relatively recent addition to the arsenal of recreational drugs of abuse. It is sold as cartridges to the catering industry for use in making whipped cream, but can be released and inhaled directly or through balloons or bags. Erroneously regarded as a safe drug it is reported to cause euphoric symptoms in users with a brief onset of actions. It is used clinically as a dental anaesthetic and anxiolytic and hence also promotes feelings of relaxation, detachment and sensory changes. Deaths have been reported due to hypoxia since catering nitrous oxide is not mixed with oxygen as it is when used medically.

Nitrous oxide can cause a rapid depletion in vitamin B12 stores in users, particularly in susceptible individuals such as vegans, anorexics, coeliac patients and those with problems with intestinal absorption. Nitrous oxide reacts with cobalt in the vitamin B12 molecule converting it from an active to an inactive bivalent form, thereby rendering it unusable in vivo. Symptoms of rapid neurological and haematological deterioration can occur including subacute combined degeneration of the cord, depression, dementia, psychosis and blindness, as well as megaloblastic anaemia. Chronic use can lead to pancytopaenia. In the acute phase, symptoms are reversible on cessation of the drug and high dose vitamin B12 supplementation. Chronic users may suffer irreversible neurological damage.

Poppers (amyl nitrate) cause euphoria and sensory dissociation on inhalation but long term effects are rare. A serious complication of their use is the oxidation of ferrous ions within haemoglobin molecules to ferric ions, creating methaemoglobinaemia, reducing the oxygen delivering capacity of the blood. They are not indicted in vitamin deficiencies.

Crack cocaine causes myriad sympathomimetic and serotonergic effects in users but is not directly attributable to anaemia or avitaminosis although this can occur secondarily in the serotonin syndrome. Crack cocaine can also cause choreoathetoid movements in some users. Similarly, heroin causes analgesic and depressant effects at opioid receptors but is not attributable directly to haematological or neurological symptoms although these may be seen in long term users due

to self neglect. Spice is a synthetic cannabinoid drug with similar but augmented effects to cannabis. CNS effects of its use include agitation, confusion, psychosis, convulsions, nystagmus and blindness. It is rare for haematological effects to occur although renal failure may be seen.

Discuss (5) | Improve

Next question >

Novel psychoactive substances

Novel psychoactive substances is the medical term for the many new substances which are chemically related to established recreational drugs such as MDMA and cannabis. They are often referred to as 'legal highs' although this is a misnomer in the UK, as their distribution and sale have been illegal since 2016.

The information below describes some of the common types:

Stimulants

- similar to MDMA, amphetamines and cocaine, resulting in increased levels of serotonin, dopamine and noradrenaline, resulting in a 'high' and feeling of euphoria
- a common example is a stimulant NPS is mephedrone ('bath salts','M-CAT'.'meow meow'). It is a cathinone and structurally similar to khat, a plant found in East Africa
- another example is benzylpiperazine ('Exodus', 'Legal X', 'Legal E')
- typically swallowed as a pill/powder ('bombing') or snorted
- adverse effect profile similar to MDMA/cocaine, with the risk of serotonin syndrome

Cannabinoids

- termed synthetic cannabinoid receptor agonists
- commonly referred to as 'spice'
- typically sprayed on to herbal mixtures which are then smoked. Also available in liquid form which is then inhaled using e-cigarettes
- similar adverse effects to cannabis

Hallucinogenics

- can be either dissociatives and psychedelics
- dissociatives produce a similar effect to ketamine, with a sense of not being connected to the physical body or time. A common dissociative NPS is methoxetamine ('mexxy')
- psychedelics have a similar effect to LSD although NPS versions may also be a stimulant

Depressant

- can be either opioid or benzodiazepine-based
- usually taken as a pill or a powder
- often structurally very similar to the original drug class, hence the adverse effects are similar
- benzodiazepine NPS often have a significantly longer half-life

Other substances include:

- Gamma-hydroxybutyric acid (GHB) and gamma-butyrolactone (GBL): 'G', 'Geebs' or 'Liquid Ecstasy'
- GHB causes respiratory depression. When taken with other respiratory depressants, most commonly alcohol, this can be potentially life threatening
- Nitrous oxide: 'Hippie crack'

For a more detailed overview please see the excellent review in BMJ 2017;356:i6848

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 42-year-old woman is admitted for dysuria, fever and rigors that have developed over the past 24 hours. A urine sample is sent to the lab and cultures demonstrate sensitivity to gentamicin. Her renal function is normal and so she is commenced on a typical loading regime.

She is stable on a twice-daily regime of gentamicin for several days. Blood tests are measured one day, pre-and post-evening dose:

Pre-dose	0.6mg/L	(<1)
One hour post-dose	7mg/L	(3-5)

What is the most appropriate action to take?

- Decrease the dose
- Decrease the time interval between doses
- Increase the time interval between doses
- Recheck levels around the next dose
- Withhold the next dose

Submit answer

Reference ranges

Score: **15.1%**

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Pre-dose	0.6mg/L	(<1)
One hour post-dose	7mg/L	(3-5)

What is the most appropriate action to take?

- Decrease the dose — 49%
- Decrease the time interval between doses — 7%
- Increase the time interval between doses — 26%
- Recheck levels around the next dose — 10%
- Withhold the next dose — 8%

For multiple daily dose regimens of gentamicin, if the pre-dose ('trough') concentration is high, the interval between doses must be increased. If the post-dose ('peak') concentration is high, the dose must be decreased

Important for me Less important

The correct answer is to decrease the dose. The post-dose ('peak') concentration is high. As such, the dose is too high and so the **dose should be reduced**.

Decreasing the time interval between doses is incorrect. This would further increase the gentamicin levels, which is inappropriate.

Increasing the time between doses is also incorrect. This would be appropriate if the pre-dose ('trough') level was too high, not the post-dose level.

Rechecking levels is not sufficient. Action needs to be taken given the result - the dose needs reducing.

Withholding the next dose would be an appropriate step if both pre-dose and post-dose levels were too high. The patient should then be rediscussed with microbiology.

Discuss (3) | Improve

Next question >

Gentamicin

Gentamicin is a type of aminoglycoside antibiotic. It is poorly lipid-soluble and is therefore given parentally (e.g. for infective endocarditis) or topically (e.g. for otitis externa).

Adverse effects

- ototoxicity
  - due to auditory or vestibular nerve damage
  - irreversible
- nephrotoxicity
  - accumulates in renal failure
  - gentamicin is preferentially taken up by the proximal renal tubular cells. Once inside these cells, it can accumulate to toxic levels
  - the toxicity is secondary to acute tubular necrosis
  - concomitant use of furosemide increases the risk
  - lower doses and more frequent monitoring is required

Contraindications

- myasthenia gravis

Dosing

- due to the significant ototoxic and nephrotoxic potential of gentamicin it is important to monitor plasma concentrations
- both peak (1 hour after administration) and trough levels (just before the next dose) are measured
- if the trough (pre-dose) level is high the interval between the doses should be increased
- if the peak (post-dose) level is high the dose should be decreased

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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You are working in the renal ward when you are asked to urgently see a 70-year-old woman. She has just returned from a tunnelled central venous catheter insertion for a planned start on dialysis and has developed tachycardia and hypotension. You note that the procedure report suggests a difficult insertion poorly tolerated by the patient. On return to the ward, she complained to the nurse of numbness around her mouth and tingling of her tongue.

She has type 2 diabetes mellitus with nephropathy and atrial fibrillation. Medication includes alfacalcidol, bisoprolol, bumetanide, and edoxaban.

Observations are as follows.

- Respiratory rate 30
- Oxygen saturations 94% on air
- Heart rate 180 beats per minute
- BP 100/65 mmHg
- T 37.4ºC

ECG shows a regular broad complex tachycardia at 180 beats per minute. An urgent blood gas shows results as follows.

Na^+	136 mmol/L	(135 - 145)
K^+	5.0 mmol/L	(3.5 - 5.0)
Glucose	6.1 mmol/L	(3.3 - 5.8)

What is the most appropriate immediate course of action?

- Administer 10% calcium gluconate and organise urgent haemodialysis
- Administer IM glucagon
- Administer IV lipid emulsion
- Commence IV antibiotics for line infection
- Urgent DC cardioversion

Submit answer

Reference ranges

Score: **15.1%**

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What is the most appropriate immediate course of action?

Administer 10% calcium gluconate and organise urgent haemodialysis	15%
Administer IM glucagon	3%
Administer IV lipid emulsion	**67%**
Commence IV antibiotics for line infection	1%
Urgent DC cardioversion	14%

Local anaesthetic toxicity can present with CNS (e.g. reduced GCS, seizures) and cardiovascular (e.g. tachyarrhythmias, hypotension, bradyarrhythmias) features. Treatment of severe toxicity is with IV lipid emulsion.

Important for me Less important

The correct answer **administer IV lipid emulsion**. This patient has likely developed local anaesthetic toxicity during the line insertion. Early clues include circumoral numbness, tongue paraesthesia, and dizziness. Patients can then develop CNS toxicity including drowsiness and coma. In this case, the patient has developed cardiovascular toxicity which can include arrhythmias such as ventricular tachycardia. The treatment of choice is IV lipid emulsion, which is thought to bind the local anaesthetic in the serum, therefore facilitating its clearance from the tissues in which it has a toxic effect.

Administer 10% calcium gluconate and organise urgent haemodialysis is incorrect. This would be the treatment for severe hyperkalaemia in a dialysis-dependent patient. While this can lead to ventricular tachycardia, the normal potassium on the blood gas is reassuring.

Administer IM glucagon is incorrect. This is a treatment option for beta-blocker overdose, which typically presents with bradycardia and resultant hypotension. Tachyarrhythmias are not a feature. It can also be used in hypoglycaemia but this would be unlikely to result in the clinical picture described. The glucose on the blood gas is also normal.

Commence IV antibiotics for line infection is incorrect. This would be very early for line infection to have developed and the lack of fever goes against this differential. Sepsis obviously could result in hypotension but would be unlikely to cause VT, which is the primary cause of the haemodynamic instability in this case.

Urgent DC cardioversion is incorrect. This would be the treatment of choice for any tachyarrhythmia with shock, defined by the resuscitation council as a systolic BP <90 mmHg. This patient's blood pressure is above this so other measures should be pursued first, particularly in the context of a toxic cause with a recognised antidote available.

👍 👎 💬 Discuss (1) Improve

Next question ›

Local anaesthetic agents ★

Lidocaine

- An amide

- Local anaesthetic and a less commonly used antiarrhythmic (affects Na channels in the axon)
- Hepatic metabolism, protein bound, renally excreted
- Toxicity: due to IV or excess administration. Increased risk if liver dysfunction or low protein states. Note acidosis causes lidocaine to detach from protein binding. Local anesthetic toxicity can be treated with IV 20% lipid emulsion
- Drug interactions: Beta blockers, ciprofloxacin, phenytoin
- Features of toxicity: Initial CNS over activity then depression as lidocaine initially blocks inhibitory pathways then blocks both inhibitory and activating pathways. Cardiac arrhythmias.
- Increased doses may be used when combined with adrenaline to limit systemic absorption.

Cocaine

- Pure cocaine is a salt, usually cocaine hydrochloride. It is supplied for local anaesthetic purposes as a paste.
- It is supplied for clinical use in concentrations of 4 and 10%. It may be applied topically to the nasal mucosa. It has a rapid onset of action and has the additional advantage of causing marked vasoconstriction.
- It is lipophillic and will readily cross the blood brain barrier. Its systemic effects also include cardiac arrhythmias and tachycardia.
- Apart from its limited use in ENT surgery it is otherwise used rarely in mainstream surgical practice.

Bupivacaine

- Bupivacaine binds to the intracellular portion of sodium channels and blocks sodium influx into nerve cells, which prevents depolarization.
- It has a much longer duration of action than lignocaine and this is of use in that it may be used for topical wound infiltration at the conclusion of surgical procedures with long duration analgesic effect.
- It is cardiotoxic and is therefore contra indicated in regional blockage in case the tourniquet fails.
- Levobupivicaine (Chirocaine) is less cardiotoxic and causes less vasodilation.

Prilocaine

- Similar mechanism of action to other local anaesthetic agents. However, it is far less cardiotoxic and is therefore the agent of choice for intravenous regional anaesthesia e.g. Biers Block.

All local anaesthetic agents dissociate in tissues and this contributes to their therapeutic effect. The dissociation constant shifts in tissues that are acidic e.g. where an abscess is present, and this reduces the efficacy.

Doses of local anaesthetics

Agent	Dose plain	Dose with adrenaline
Lignocaine	3mg/Kg	7mg/Kg
Bupivacaine	2mg/Kg	2mg/Kg
Prilocaine	6mg/Kg	9mg/Kg

These are a guide only as actual doses depend on site of administration, tissue vascularity and co-morbidities.

Maximum total local anaesthetic doses

- Lignocaine 1% plain - 3mg/ Kg - 200mg (20ml)
- Lignocaine 1% with 1 in 200,000 adrenaline - 7mg/Kg - 500mg (50ml)
- Bupivicaine 0.5% - 2mg/kg- 150mg (30ml)

Maximum doses are based on ideal body weight

Effects of adrenaline

Adrenaline may be added to local anaesthetic drugs. It prolongs the duration of action at the site of injection and permits usage of higher doses (see above). It is contra indicated in patients taking MAOI's or tricyclic antidepressants. The toxicity of bupivacaine is related to protein binding and addition of adrenaline to this drug does not permit increases in the total dose of bupivacaine, in contrast to the situation with lignocaine.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Local anesthetics

Osmosis - YouTube 6 0

General & local anaesthetics

Speed Pharmacology - YouTube 2 0

Local anaesthetic toxicity

Kloss and Bruce - YouTube 4 1

Report broken media

Score: **18.1%**

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A 48-year-old man is reviewed in the Medical Admissions unit complaining of palpitations. He has a past medical history of hereditary obstructive cardiomyopathy (HOCM), for which he has taken amiodarone for the last year. He has no personal or family history of autoimmune disease.

On examination, he is flushed and diaphoretic. He appears thin, and he has a fine tremor when his hands are outstretched. There is no discernible goitre.

His observations are as follows: heart rate 102/min; blood pressure 126/82 mmHg; respiratory rate 14/min; temperature 37.9ºC; oxygen saturations 97% on air.

Selected blood test results are shown below:

Free T4	28.6 pmol/L	(12 - 22)
Free T3	9.1 pmol/L	(3.1 - 6.8)
TSH	< 0.05 mU/L	(0.27 - 4.2)

Given the likely diagnosis, what is the most appropriate management option?

- Carbimazole
- Lithium
- Lugol's iodine
- Prednisolone
- Propylthiouracil

Submit answer

Reference ranges

Score: **15.1%**

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TSH	< 0.05 mU/L	(0.27 - 4.2)

Given the likely diagnosis, what is the most appropriate management option?

Carbimazole	17%
Lithium	1%
Lugol's iodine	4%
Prednisolone	72%
Propylthiouracil	7%

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

Important for me Less important

The correct answer is **prednisolone**. This patient has a likely diagnosis of amiodarone-induced thyrotoxicosis (AIT) type 2. The thyroid function tests are in keeping with thyroid overactivity, whilst the examination findings clearly describe him as clinically thyrotoxic. In the absence of an alternative explanation (e.g. known Graves disease), this is most likely due to the patient taking

amiodarone. The diagnosis of AIT type 2 is more likely than AIT type 1, as AIT type 2 is a destructive thyroiditis that tends not to present with goitre formation. The initial management of AIT type 2 is with corticosteroids such as prednisolone.

Carbimazole is incorrect. This would be an appropriate initial management option for this patient if he had a goitre on examination, and hence if the clinical picture were more consistent with AIT type 1. Antithyroid drugs are favoured in AIT type 1, where the pathophysiology involves excess thyroid hormone synthesis stimulated by the
iodide load from amiodarone therapy (the Jod-Basedow phenomenon).

Lithium is incorrect. This would be an appropriate management option for either AIT type 1 or type 2 that was refractory to initial therapy (with either antithyroid drugs or corticosteroids respectively).

Lugol's iodine is incorrect. Lugol's iodine is used for its antithyroid properties in the management of severe thyrotoxicosis, and as a preparation for thyroidectomy surgery in patients with Graves disease. It is not routinely used in the management of AIT type 2, where the anti-inflammatory properties of corticosteroids are preferred.

Propylthiouracil is incorrect as corticosteroids, rather than antithyroid drugs are the first-line management option for AIT type 2.

Discuss (1) | Improve

Next question >

Amiodarone and the thyroid gland ★

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	AIT type 2
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

Score: **18.1%**

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You are called to the emergency department to review a 33-year-old man. He is an Estonian national and works on a farm. His spoken English is poor but he tells you he feels unwell. He is being treated in a side-room because of loose stools.

On examination he is drooling slightly and has watery eyes. His pupils are equal in size but miotic. His blood pressure is 102/58 mmHg and an ECG shows sinus bradycardia.

How should this patient be managed? :

- Amiodarone
- Pyridostigmine
- Glucagon
- Haemodialysis
- Atropine

Submit answer

Reference ranges

Score: **15.1%**

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On examination he is drooling slightly and has watery eyes. His pupils are equal in size but miotic. His blood pressure is 102/58 mmHg and an ECG shows sinus bradycardia.

How should this patient be managed? :

Amiodarone	0%
Pyridostigmine	8%
Glucagon	4%
Haemodialysis	2%
Atropine	86%

Organophosphate insecticide - atropine

Important for me Less important

This man has features of organophosphate poisoning and demonstrates some of the anticholinesterase effects exerted by industrial insecticides. The only suitable treatment listed is atropine. Atropine exerts an anticholinergic effect at the level of the muscarinic receptor, thus opposing the effects of accumulated acetylcholine.

Amiodarone is not typically used to treat bradyarrhythmias. Glucagon can be used in beta-blocker overdose, but beta blockade would not cause the miotic pupils or lacrimation. Pyridostigmine is an anticholinesterase agent used in the treatment of myasthenia gravis; it would do nothing to help in this situation.
Organophosphates are not amenable to haemodialysis (remember the 'BLAST' mnemonic for overdoses that can be dialysed:
Barbiturates
Lithium
Alcohols e.g. ethylene glycol

Salicylates
Theophyllines)

Discuss (3) Improve

Next question

Organophosphate insecticide poisoning

One of the effects of organophosphate poisoning is inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission. In warfare, sarin gas is a highly toxic synthetic organophosphorus compound that has similar effects.

Features can be predicted by the accumulation of acetylcholine (mnemonic = SLUD)

- Salivation
- Lacrimation
- Urination
- Defecation/diarrhoea
- cardiovascular: hypotension, bradycardia
- also: small pupils, muscle fasciculation

Management

- atropine
- the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit

123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 17-year-old woman with a history of epilepsy and deliberate self-harm is brought into the emergency department with a Glasgow coma score of 13 (E3 V4 M6) and respiratory rate of 8/min.

On examination, her pulse is 56/min and regular, blood pressure 110/60 mmHg and her chest is clear. She has no signs of injury but an empty packet of diazepam was found in her handbag. Whilst the patient is breathing room air a nurse in the emergency department has taken an arterial blood gas shown below.

pH	7.39
pO2	10.1 kPa
pCO2	5.6 kPa
BE	0.8 mEq/l

What is the best initial management?

- Flumazenil 200mcg infused IV over 15 minutes
- Flumazenil 200mcg bolus IV over 15 seconds
- Naloxone 400mcg IM injection
- Urgent intubation with anaesthetic support
- Supportive care only

Submit answer

Reference ranges

Score: **15.1%**

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On examination, her pulse is 56/min and regular, blood pressure 110/60 mmHg and her chest is clear. She has no signs of injury but an empty packet of diazepam was found in her handbag. Whilst the patient is breathing room air a nurse in the emergency department has taken an arterial blood gas shown below.

pH	7.39
pO2	10.1 kPa
pCO2	5.6 kPa
BE	0.8 mEq/l

What is the best initial management?

Flumazenil 200mcg infused IV over 15 minutes	27%
Flumazenil 200mcg bolus IV over 15 seconds	14%
Naloxone 400mcg IM injection	5%
Urgent intubation with anaesthetic support	20%
Supportive care only	34%

The blood gas shows her ventilation appears inadequate but at this stage the best management is supportive, allowing levels of diazepam to reduce over time, something that can be easily assessed clinically with and with the aid of an arterial blood gas sample.

Administering the benzodiazepine antidote flumazenil to an epileptic puts her at high risk of seizures. Naloxone is an antidote to opiate overdose. If however despite simple manoeuvres her ventilation deteriorated or GCS dropped to less than 8, anaesthetic support should be sought.

Overdose and poisoning: management ★

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization with IV bicarbonate • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil The majority of overdoses are managed with supportive care only due to the risk of seizures with flumazenil. It is generally only used with severe or iatrogenic overdoses.
Tricyclic antidepressants	Management • IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity • arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics
Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes

Toxin	Treatment
	lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine • in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit
Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen

Toxin	Treatment
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

Emergency treatment of poisoning

2 1

Royal College of Physicians

2012 Poisoning and self-harm update

1 1

Royal College of Physicians

2007 Acute poisoning on the medical admissions unit

1 2

Suggest link

Report broken link

Score: **18.1%**

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A 55-year-old woman presents to the emergency department with shortness of breath. She has no significant past medical history and does not have any regular medications. She smokes ten cigarettes daily and lives alone.

Her observations are heart rate 110 beats per minute, blood pressure 120/88 mmHg, respiratory rate 18/minute, oxygen saturations 97% on 4L of oxygen and temperature 37ºC.

Clinical examination is unremarkable.

Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	180 * 10^9/L	(150 - 400)
WBC	5.2 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
D-dimer	2000 ng/mL	(<250)
Trop	8 ng/L	(<15)

A chest x-ray is normal.

A CTPA demonstrates a large saddle embolus. There is no CT or echocardiographic evidence of right heart strain.

She is commenced on a heparin infusion. She successfully manages to wean oxygen by day 5 of treatment and has some repeat blood tests prior to a switch to an oral anticoagulant.

Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)

Platelets	32 * 10^9/L	(150 - 400)
WBC	4.6 * 10^9/L	(4.0 - 11.0)

Given the likely diagnosis, what is the most suitable medication to switch to from the heparin infusion?

- ○ Abciximab ×
- ○ Argatroban ×
- ○ Enoxaparin ×
- ○ Platelet transfusion ×
- ○ Warfarin ×

Submit answer

Reference ranges

Score: **15.1%**

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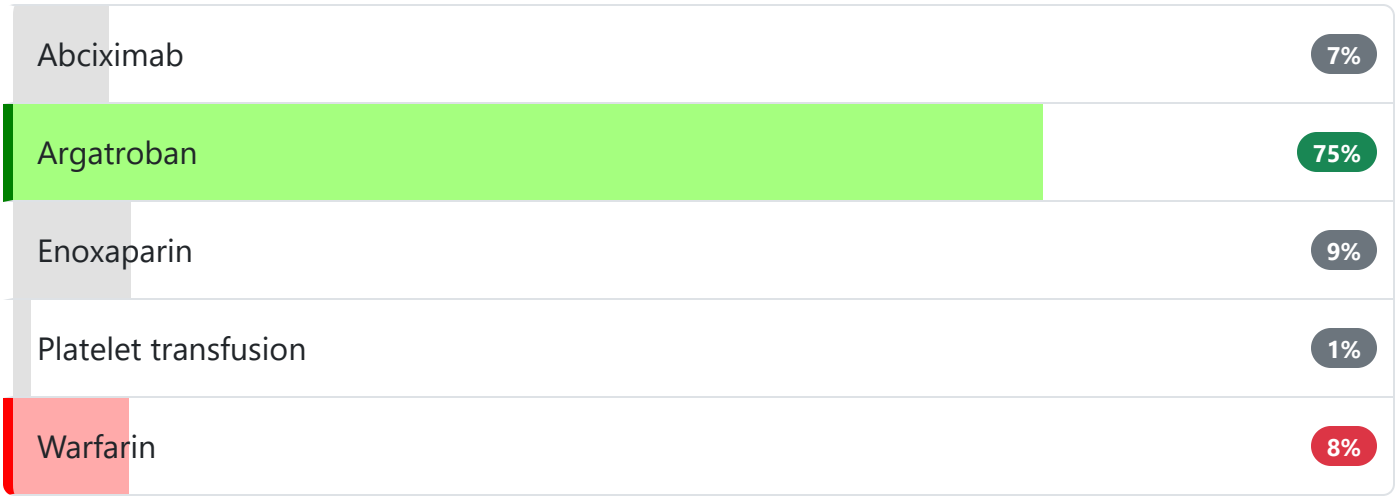

Heparin-induced thrombocytopenia: anticoagulation can be provided by direct thrombin inhibitor e.g. argatroban

Important for me Less important

Argatroban is the correct answer. The patient has heparin-induced thrombocytopenia, as evidenced by a > 50% drop in platelets with a nadir platelet count > 20 * 10^9/L, occurring between days 5-10 post heparin administration, in the absence of another cause. She does not have any clinical evidence of thrombosis clinically but is at high risk of thrombotic events. Argatroban is a small molecule direct thrombin inhibitor and it is given intravenously. Patients can be transitioned to an oral anticoagulant once the platelet count has recovered.

Abciximab is incorrect. This is a glycoprotein IIb/IIIa receptor antagonist and is used as an antiplatelet medication rather than an anticoagulant.

Enoxaparin is incorrect. This is a rarer cause of induced-thrombocytopenia but is not recommended in the management of this condition.

Platelet transfusion is incorrect. The patient is not bleeding and the platelet count is > 30. Therefore platelets are not indicated. Indeed, they may increase thrombotic risk.

Warfarin is incorrect. The patient can be transitioned from argatroban to warfarin once the platelet count has recovered.

Heparin

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as	Now standard in the management of venous thromboembolism

	Standard heparin	Low molecular weight heparin (LMWH)
	anticoagulation can be terminated rapidly. Also useful in renal failure	treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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Sonia is a 24-year-old woman presenting to the emergency department after being brought in by her flatmate. Sonia was found collapsed on the ground surrounded by unmarked bottles. She was noted to have been arguing with her boyfriend earlier today and given her history of depression, her friend thought she should have checked to ensure Sonia was safe. She is unsure is the bottles contained medications or other liquids.

Sonia was initially significantly disorientated on presentation and could not accurately recall where she was or why she was in the hospital. She began to experience nausea and vomiting while in the emergency department. After 1 hour in the emergency department, she became incredibly drowsy and became unrousable.

Physical examination was otherwise unremarkable. Skin colour was relatively normal. There was no tenderness on palpation of her abdomen. She is tachypnoeic with a respiratory rate of 30 breaths/minute.

Blood tests are as follow:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	250 * 10^9/L	(150 - 400)
WBC	7.0 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	5.0 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)
CRP	<5 mg/L	(< 5)
Bicarbonate	12 mmol/L	(22 - 29)
Chloride	98mmol/L	(95-105)
Measured osmolarity	350mOsm/kg	(275-295)
Bilirubin	14 µmol/L	(3 - 17)
ALP	60 u/L	(30 - 100)
ALT	30 u/L	(3 - 40)
Î³GT	40 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

Arterial blood gases showed:

pH	7.27	7.35-7.45
pO2	13.7 kPa	11-13 kPa
pCO2	3.2 kPa	4.7 - 6.0kPa
HCO3	12	22-26
BE	-4	-2 - +2

An ECG was performed and this showed sinus tachycardia. No abnormalities of the QRS complex was noted.

What is the likely agent that had been taken?

- ○ Aspirin ×
- ○ Ethylene glycol ×
- ○ Iron tablets ×
- ○ Organophosphate pesticide ×
- ○ Paracetamol ×

Submit answer

Reference ranges

Score: **15.1%**

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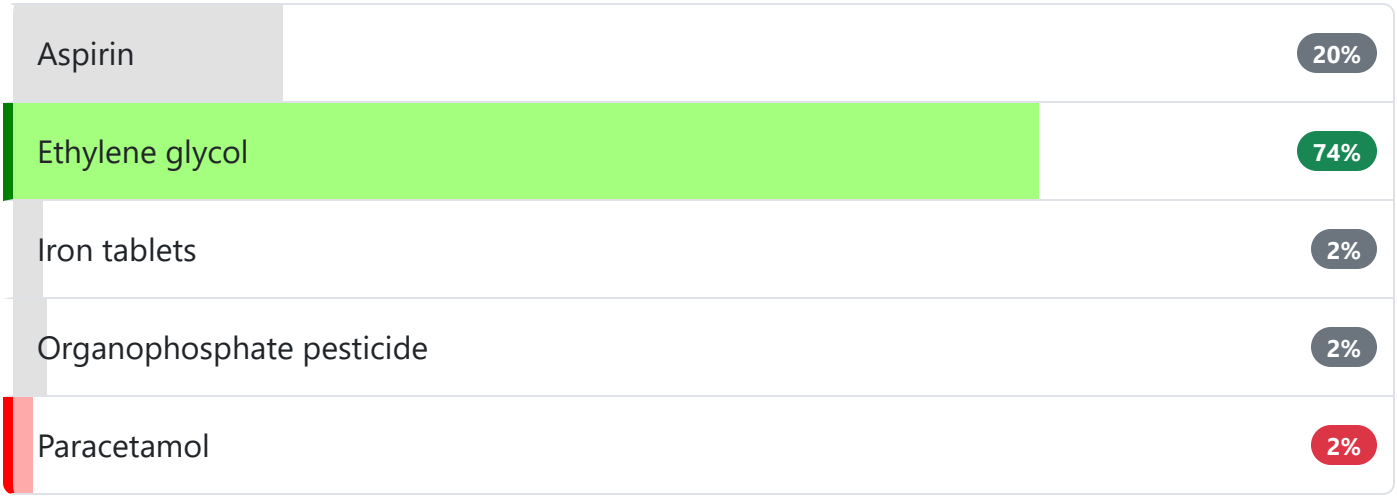

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

The patient has taken an overdose of an unknown substance. Her only symptoms included nausea, vomiting before progressing to drowsiness and stupor, all relatively non-specific.

Her blood tests do provide additions information as this shows a metabolic acidosis from her arterial blood gas. Further assessment of her blood tests reveals an underlying raised anion gap along with a raised osmolar gap. Osmolar gap is calculated by measured serum osmolarity - calculated serum osmolarity (2x Na^+ + glucose + urea). A gap of >=10 suggests an elevated osmolar gap.

Causes of a raised osmolar gap are alcohol ingestion (including methanol and ethylene glycol), sugars (mannitol and sorbitol), lipids (hypertriglyceridemia) and proteins (hypergammaglobulinemia). Given the combination of her blood tests and symptoms, ethylene

glycol remains the likely substance ingested.

Aspirin can cause metabolic acidosis with a raised anion gap. However, it will be associated with more symptoms including tinnitus and fevers. Also, it is not associated with a raised osmolar gap.

Iron overdose will present with other symptoms including haematemesis and abdominal pains. It can present with a raised anion gap metabolic acidosis.

Paracetamol overdose will not result in the blood tests seen. Also, it can present with hepatic dysfunction before progression to hepatic encephalopathy in severe cases.

Organophosphate poisoning is unlikely as the patient does not display any clear cholinergic symptoms including salivation, lacrimation or diarrhoea.

Discuss (7) Improve

Next question

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **18.1%**

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A 32-year-old mechanic is admitted to the emergency department. He is drowsy and confused. He is accompanied by his girlfriend who informs you they had been drinking alcohol the previous night. She became concerned when he appeared increasingly confused throughout the day despite no further alcohol intake. She tells you his mood has been low over the last number of months and has recently expressed suicidal ideation.

On your assessment his Glasgow coma scale is 14/15, pupils are equal and reactive to light. His Blood pressure is 124/81mmHg with a heart rate of 97/minute. Oxygen saturations are 94% on room air and he is tachypnoeic with a respiratory rate of 32/minute. There is bilateral flank tenderness, the clinical exam is otherwise unremarkable.

His urine toxicology screen is clear. Urine dipstick is positive for blood.

Arterial blood gas:

PH	7.10
PO2	12.3Kpa
PCO2	2.6Kpa
Bicarbonate	8.0mEq/L
Lactate	2.3mmol/L

Biochemistry:

Na^+	145 mmol/L	(135 - 145)
K^+	3.8 mmol/L	(3.5 - 5.0)
Cl^+	105 mmol/L	(98-106)
Urea	11mmol/L	(2.0 - 7.0)
Creatinine	113µmol/L	(55 - 120)
Glucose	5.7mmol/L	(3.9-5.8)
Serum Osmolality	358mOsm/Kg	(275-295)
Ethanol	<10mmol/L	
Paracetamol	0mg/dl	

Which of the following is the most likely cause for this man's presentation?

- ○ Alcoholic ketoacidosis ×
- ○ Ethylene glycol toxicity ×
- ○ Alcohol intoxication ×
- ○ Methanol poisoning ×
- ○ Paracetamol overdose ×

Submit answer

Reference ranges

Score: **15.1%**

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Serum Osmolality	358mOsm/Kg	(275-295)
Ethanol	<10mmol/L	
Paracetamol	0mg/dl	

Which of the following is the most likely cause for this man's presentation?

Alcoholic ketoacidosis	5%
Ethylene glycol toxicity	78%
Alcohol intoxication	2%
Methanol poisoning	15%
Paracetamol overdose	0%

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

This question requires you to identify a metabolic acidosis with a high anion gap and a high osmolar gap. This can be seen due to the presence of several different compounds or their toxic metabolites (glycols, methanol, oxoproline, lactate, ketones, aspirin, etc). In this case, the clinical history allows you to identify the likely cause.

Ethylene glycol and methanol are commonly found in automotive coolant, antifreeze, windshield wiper fluid, solvents, cleaners, and fuels. This patient is a mechanic with easy access to these compounds. Both methanol and ethylene glycol are metabolized by alcohol dehydrogenase (ADH) before having their effects via toxic metabolites. Ethanol competitively inhibits ADH, thereby diminishing the formation of toxic metabolites of the parent compound. Our patient had been drinking alcohol the previous night which may have masked his early symptoms and delayed the formation of toxic metabolites.

The clinical findings can help differentiate between methanol and ethylene glycol toxicity. Advanced methanol toxicity is associated with retinal injury and visual disturbance. Ethylene glycol more commonly presents with oliguria, hematuria, and flank pain. This patient had microscopic haematuria and bilateral flank pain. Definitive diagnosis can be made with direct testing of toxic alcohols or by identifying the consumed substance.

Calculating the anion gap:

Anion gap mmol/L = sodium mmol/L - (chloride mmol/L + bicarbonate mmol/L).
Calculated anion gap = 32 (normal range 8->12).

Calculating the osmolar gap:

Osmolal gap = measured serum osm (358) - calculated serum osm (307).
Serum osmolality = (2 Ã— Na + (BUN / 2.8) + (glucose / 18) + (ethanol / 3.7).
Calculated serum osmolality = 307 mOsm/kg.
Measured serum osmolality = 358mOsm/kg.
Osmolal gap = 51(normal range (-10) - (+10).

Discuss (3) Improve

Next question

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

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AI + Q 123

Next question

B I A T

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **18.1%**

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A 47-year-old Latvian man is brought to the Emergency Department by two of his friends. He is confused and aggressive and walks with an unsteady gait. History is difficult to obtain from the patient due to slurring of his speech and a slight language barrier. You note that he fails to meet your gaze when you are talking to him. Examination shows a GCS of 13/15 but he is drowsy and irritable. There is a vague smell of alcohol on his breath. He is flushed in the face and his eyes are red and hyperaemic. Pupils are slow to react to direct light but equal in size. The cardiorespiratory examination is unremarkable aside from a respiratory rate of only 8/min but SpO2 is 98% on air. The abdomen is soft and non-tender with no hepatomegaly and no signs of chronic liver disease. Despite the department being well lit, he asks you to turn the lights on.

Biochemistry reveals:

Haemoglobin	149g/L	Sodium	150mmol/L	pH	7.25
White cells	$8.9x10^9$/L	Potassium	5.9mmol/L	pC02	3.9kPa
Neutrophils	$5.9x10^9$/L	Urea	13.8mmol/L	pO2	12.6kPa (air)
Platelets	$229x10^9$/L	Creatinine	156mol/L	HCO3-	12.5mmol/L
Prothrombin time	11 sec	Glucose	24.7mmol/L	BE	-13.2mEq/L
Ethanol	110mg/dL	Chloride	100mmol/L	Lactate	2.0mmol/L
Anion gap	38	Osmolar gap	25 (-10 to +15)		

What is the most appropriate next step in the management of this patient?

- Begin high dose oral folic acid and vitamin B complex
- Establish IV access and begin fast fluids with Pabrinex I+II (Vitamin B12)
- Establish IV access and send blood for methanol levels; give oral ethanol in the form of neat 40% whisky at a dose of 175ml
- Establish IV access and send blood for methanol levels; begin IV fomepizole 1000mg in 500ml 5% dextrose over 30 minutes
- Refer to Intensive Care to begin haemofiltration

Submit answer

Reference ranges

Score: **15.1%**

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Ethanol	110mg/dL	Chloride	100mmol/L	Lactate	2.0mmol/L
Anion gap	38	Osmolar gap	25 (-10 to +15)		

What is the most appropriate next step in the management of this patient?

Begin high dose oral folic acid and vitamin B complex	2%
Establish IV access and begin fast fluids with Pabrinex I+II (Vitamin B12)	5%
Establish IV access and send blood for methanol levels; give oral ethanol in the form of neat 40% whisky at a dose of 175ml	4%
Establish IV access and send blood for methanol levels; begin IV fomepizole 1000mg in 500ml 5% dextrose over 30 minutes	**85%**
Refer to Intensive Care to begin haemofiltration	3%

Methanol poisoning is preferentially treated with IV fomepizole; this should be commenced immediately the suspicion is raised

Important for me Less important

The clinical picture in this scenario is typical of an acute alcohol intoxication syndrome with descriptors of ataxia, confusion, aggression and drowsiness. Often there will be a smell of alcohol present when examining the patient and unfortunately, a coherent history is often absent due to poor compliance and often incapacity. However, in the vignette, there are features which might not be expected with a case of ethanol intoxication such as difficulties with vision, often detected by the failure of making any eye contact or of requesting extra lighting in a well-lit room. This suggests damage to the optic nerve and is a feature of methanol poisoning. Methanol toxicity is often very difficult to distinguish from ethanol intoxication but visual symptoms should prompt consideration coupled with a careful history.

Similar to drinkable ethanol, methanol is rapidly metabolised by hepatic enzymes; ethanol dehydrogenase converts methanol to formaldehyde and aldehyde dehydrogenase further metabolises this to formic acid (formate) which is extremely toxic to the central nervous system. Accumulation of formate in the central nervous system leads not only to cellular hypoxia due to interruption of mitochondrial cytochrome activity but also a profound acidosis. Coupled with this methanol itself, similar to ethanol, causes central nervous and respiratory depression further augmenting its toxicity.

Typically bloods will show a metabolic acidosis with a high anion gap - often co-ingestion of ethanol will have occurred and this may account for some of the unmeasured ions but often the plasma ethanol level will not be sufficiently raised to explain it in totality. Calculation of the osmolar gap may also be elevated initially which is not seen so much with ethanol toxicity.

Treatment of methanol toxicity is by blockade of the metabolism of methanol to formate leading to renal clearance of the unmetabolised methanol. Both ethanol and fomepizole are competitive inhibitors of methanol metabolism as they preferentially have an affinity for both ethanol dehydrogenase and aldehyde dehydrogenase and so toxic formate is not produced. Fomepizole, although very expensive, is preferentially used over ethanol as there is a risk of precipitating ethanol toxicity with its associated central nervous and respiratory depression. If ethanol is used as an antidote, regular monitoring of plasma ethanol concentrations are needed to ensure maintenance within the range of 100-200mg/dL. In this case, plasma ethanol levels are within this range anyway and methanol toxicity is still occurring. Both fomepizole and ethanol need to be continued until plasma methanol concentrations fall below <50mg/L.

In addition, intravenous folinic acid should be given as this aids in the speedy metabolism of formic acid and helps in reducing the central nervous toxicity. Unfortunately, oral folic acid is insufficiently rapidly absorbed to be helpful in this situation. Supplementation of B vitamins is important in cases of any alcohol toxicity as this avoids development of Wernicke's encephalopathy and this should take the form of Pabrinex I+II, however, it is not the most pressing

concern in a potential methanol toxicity case. Oral vitamin B complex is important in chronic alcoholism but not in an acute situation. Haemofiltration is a potential mechanism for clearing methanol from the circulation, however, this is usually reserved for occasions when fomepizole fails or is not tolerated.

In cases of methanol toxicity, an intensive care opinion may be useful however as central venous access may be required to administer bicarbonate in severe acidosis or in case of respiratory depression requiring intubation and assisted ventilation.

Note that in cases of suspected methanol poisoning, commencing an antidote immediately is important and should be done before methanol levels have been confirmed.

Discuss (4) | Improve

Next question ›

Methanol poisoning ★

Methanol poisoning causes both the effects associated with alcohol (intoxication, nausea etc) and also specific visual problems, including blindness. These effects are thought to be secondary to the accumulation of formic acid. The actual pathophysiology of methanol-associated visual loss is not fully understood but it is thought to be caused by a form of optic neuropathy

Management

- fomepizole (competitive inhibitor of alcohol dehydrogenase) or ethanol
- haemodialysis
- cofactor therapy with folinic acid to reduce ophthalmological complications

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

1 5

Suggest link

Report broken link

Score: **18.1%**

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A 30-year-old man was referred to endocrinology clinic for the assessment of the adverse consequences of anabolic steroid use. The patient reported that he had recently decided to stop using anabolic steroids, having been a regular user for the previous 5 years to support his body-building training. The patient cited the potential health risks of long-term anabolic steroid use as the reason behind his decision to cease his use.

The patient had followed a regime of an intramuscular injection of long-acting synthetic testosterone derivative every two weeks. The patient took a 4-week break from anabolic steroids every 12 weeks, in an attempt to limit the adverse side-effects. The patient denied ever having used oral or topical synthetic testosterone preparations and had always used sterile injecting equipment.

The patient stated that he had developed significant gynaecomastia and also suffered significant male pattern baldness since starting to use anabolic steroids. In addition, 2 years previously he had suffered a ruptured right biceps tendon while exercising, requiring a surgical repair and had a prolonged period of rehabilitation. There was no history of symptoms suggestive of cardiac or liver disease. The patient took no regular prescribed medications and reported an alcohol consumption of between 15 to 20 units per week.

General examination of the patient revealed a highly muscular and lean adult male. Moderate gynaecomastia was present, but there were no other signs of chronic liver disease. Examination of the cardiovascular and respiratory systems was unremarkable.

Please see below for the available results of blood tests taken prior to the patient's attendance at the clinic.

HbA1C	47 mmol / mol (reference < 42)
Total cholesterol	6.1 mmol / L (reference < 5.0)
Fasting LDL cholesterol	5.0 mmol / L (reference < 3.0)
Fasting HDL cholesterol	1.1 mmol / L (reference > 1.2)
Prolactin	490 mU / L (reference 80 - 400)
Luteinising hormone	*result pending*
Follicle-stimulating hormone	*result pending*
Testosterone	*result pending*
Epitestosterone	*result pending*

What pattern of results for the pending blood tests is consistent with the patient's anabolic steroid use?

- Elevated testosterone:epitestosterone ratio; normal luteinising hormone; normal follicle-stimulating hormone
- Suppressed testosterone:epitestosterone ratio; elevated luteinising hormone; suppressed follicle-stimulating hormone
- Suppressed testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressed follicle-stimulating hormone
- Elevated testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressed follicle-stimulating hormone
- Normal testosterone:epitestosterone ratio; elevated luteinising hormone; elevated follicle-stimulating hormone

Submit answer

Reference ranges

Score: **15.1%**

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Prolactin	490 mU / L (reference 80 - 400)
Luteinising hormone	*result pending*
Follicle-stimulating hormone	*result pending*
Testosterone	*result pending*
Epitestosterone	*result pending*

What pattern of results for the pending blood tests is consistent with the patient's anabolic steroid use?

Elevated testosterone:epitestosterone ratio; normal luteinising hormone; normal follicle-stimulating hormone	3%
Suppressed testosterone:epitestosterone ratio; elevated luteinising hormone; suppressed follicle-stimulating hormone	12%
Suppressed testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressed follicle-stimulating hormone	25%
Elevated testosterone:epitestosterone ratio; suppressed luteinising hormone; suppressed follicle-stimulating hormone	57%
Normal testosterone:epitestosterone ratio; elevated luteinising hormone; elevated follicle-stimulating hormone	3%

The patient has developed some of the typical unwanted effects associated with anabolic steroid use; namely gynaecomastia and accelerated male pattern baldness. In addition, tendon rupture is frequently associated with anabolic steroid use due to the disproportionate enhancement of muscle bulk compared to tendon strength. Other common unwanted effects include acne, testicular atrophy (or clitoral hypertrophy in females), impaired glucose tolerance and a deranged blood lipid profile. More worryingly, anabolic steroid use is also associated with serious liver disease and an increased mortality from heart disease.

Epitestosterone is a naturally occurring stereoisomer of testosterone produced by the testes. In healthy males, the testosterone:epitestosterone ratio is typically 1:1. However, exogenous administration of testosterone does not increase levels of epitestosterone, so anabolic steroid use is associated with an increase in the testosterone:epitestosterone ratio (typically to greater than 4:1). In anti-doping testing for competitive sports, an elevated testosterone:epitestosterone ratio is considered possible evidence of anabolic steroid use.

Physiological production of testosterone is promoted by the release of the gonadotropins luteinising hormone and follicle stimulating hormone by the pituitary gland. Both these hormones increase the number of testosterone-producing Leydig cells in the testes, with luteinising hormone also increasing testosterone production by Leydig cells. Elevated testosterone levels reduce gonadotropin production via negative feedback to the hypothalamus (causing reduced gonadotropin releasing hormone production) and the pituitary gland. Therefore, high levels of exogenous testosterone will act to suppress luteinising hormone and follicle stimulating hormone.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

Anabolic steroid use

Anabolic steroid use is associated with several serious long-term health consequences. Cardiac morbidity and mortality are increased by anabolic steroid use, although the precise mechanism of this effect is unclear. Hepatic side effects also occur secondary to chronic vascular injury: these include hepatocellular carcinoma and hepatic adenoma. Psychiatric illness is also commonly co-morbid with anabolic steroid use. Additionally, users who inject anabolic steroids have an increased risk of blood-borne viruses if needles are shared between individuals.

Due to the above concerns, patients should be strongly counselled to stop using anabolic steroids. There is no requirement for tapering of doses. Many of the above blood test abnormalities normalise once anabolic steroid consumption ceases. The expert recommendation is for lifelong monitoring for potential complications, initially annually with frequency reducing once blood markers normalise and in the absence of apparent adverse effects.

Brooks J, Ahmad I, Easton G. Anabolic steroid use. BMJ 2016;353:i5023.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 64-year-old man presents with several months of weight loss, anxiety, and palpitations. His wife mentions that he keeps the house temperature cool as he is generally feeling too hot. He has a background of ventricular tachycardia, for which he has been taking amiodarone for some years.

On examination, he has a slight tremor and appears thin. His heart rate is 105/min. There is no goitre present.

Blood tests are taken:

Thyroid stimulating hormone (TSH)	<0.05 mU/L	(0.5-5.5)
Free thyroxine (T4)	25 pmol/L	(9.0 - 18)

He is subsequently referred for an ultrasound scan of the neck, which shows absent vascularity and probable gland destruction.

What is the most appropriate treatment?

- Carbimazole
- Iodine
- Prednisolone
- Propranolol
- Propylthiouracil

Submit answer

Reference ranges

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Free thyroxine (T4)	25 pmol/L	(9.0 - 18)

He is subsequently referred for an ultrasound scan of the neck, which shows absent vascularity and probable gland destruction.

What is the most appropriate treatment?

Carbimazole	11%
Iodine	1%
Prednisolone	79%
Propranolol	5%
Propylthiouracil	4%

In amiodarone induced thyrotoxicosis type 2 the management is with corticosteroids rather than carbimazole

Important for me Less important

This patient is likely experiencing amiodarone-induced thyrotoxicosis (AIT). This can be divided into two types. Type 1 results from excess iodine-induced thyroid hormone synthesis; a goitre is typically present. Type 2 results from amiodarone-related destructive thyroiditis and, as in this scenario, a goitre is absent. Differentiation of the two types is important as they have different management. The ultrasound scan here also shows probable gland destruction, pointing towards

type 2. Management involves the use of corticosteroids and so **prednisolone** is correct.

Carbimazole is typically used for the treatment of hyperthyroidism and would be suitable for the management of type 1 AIT. However, this scenario is more likely to be occurring as a result of type 2 AIT and so prednisolone remains more appropriate.

Iodine may be used as part of the management of hyperthyroidism generally, as well as a thyroid storm. Definitive management for type 2 AIT, however, is corticosteroid use.

Propranolol is a beta-blocker and can be used for short-term symptom relief of hyperthyroidism. It does not play a role in the definitive management of type 2 AIT.

Propylthiouracil is another drug used in the management of hyperthyroidism. Again, with the management of type 2 AIT specifically, corticosteroids are more appropriate.

Discuss (1) | Improve

Next question >

Amiodarone and the thyroid gland ★

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent

	AIT type 1	AIT type 2
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

Score: **18.1%**

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A 45-year-old man was found unconscious at home by his partner, with empty packs of flecainide, codeine and valerian root tablets besides him. He was only found after his partner got worried when he had locked himself in his room and after 4 hours not heard any sounds from the room. He had been depressed for the past couple of months and relations between him and his partner had been strained. He has a past medical history of back pain, paroxysmal atrial fibrillation, angina and hypertension.

On examination, he was drowsy, unable to give a history, with a heart rate of 50bpm, respiratory rate of 16 breaths per minute, oxygen saturation of 96% on air, and a blood pressure of 110/58mmHg. Pupils were 3mm and equal and reactive to light.

Investigations:

Na+	136 mmol/l
K+	4.6 mmol/l
Urea	10.9 mmol/l
Creatinine	110 µmol/l
Serum bilirubin	30 µmol/l
Serum alkaline phosphatase	135 IU/l
Serum aspartate aminotransferase	50 IU/l
C Reactive protein	2mg/l
Haemoglobin	14.6 g/dl
White cell count	5.6 x 109/L
INR	1.4

ABG (on air):

pH	7.258
pO2	11.7 kPa
pCO2	3.4 kPa
Lactate	1.6 mmol/l
Base Excess	-8.4 mmol/l
Bicarbonate	11.9 mmol/l

ECG showed bradycardia with widened QRS complexes and giant inverted T waves.

After fluid resuscitation, what is the next single most important management step?

- ○ Activated Charcoal ×
- ○ Magnesium Sulphate ×
- ○ Sodium Bicarbonate ×
- ○ Haemodialysis ×
- ○ N-Acetyl Cysteine infusion (NAC) ×

Submit answer

Reference ranges

Score: **15.1%**

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A 45-year-old man was found unconscious at home by his partner, with empty packs of flecainide, codeine and valerian root tablets besides him. He was only found after his partner got worried when he had locked himself in his room and after 4 hours not heard any sounds from the room. He had been depressed for the past couple of months and relations between him and his partner had been strained. He has a past medical history of back pain, paroxysmal atrial fibrillation, angina and hypertension.

On examination, he was drowsy, unable to give a history, with a heart rate of 50bpm, respiratory rate of 16 breaths per minute, oxygen saturation of 96% on air, and a blood pressure of 110/58mmHg. Pupils were 3mm and equal and reactive to light.

Investigations:

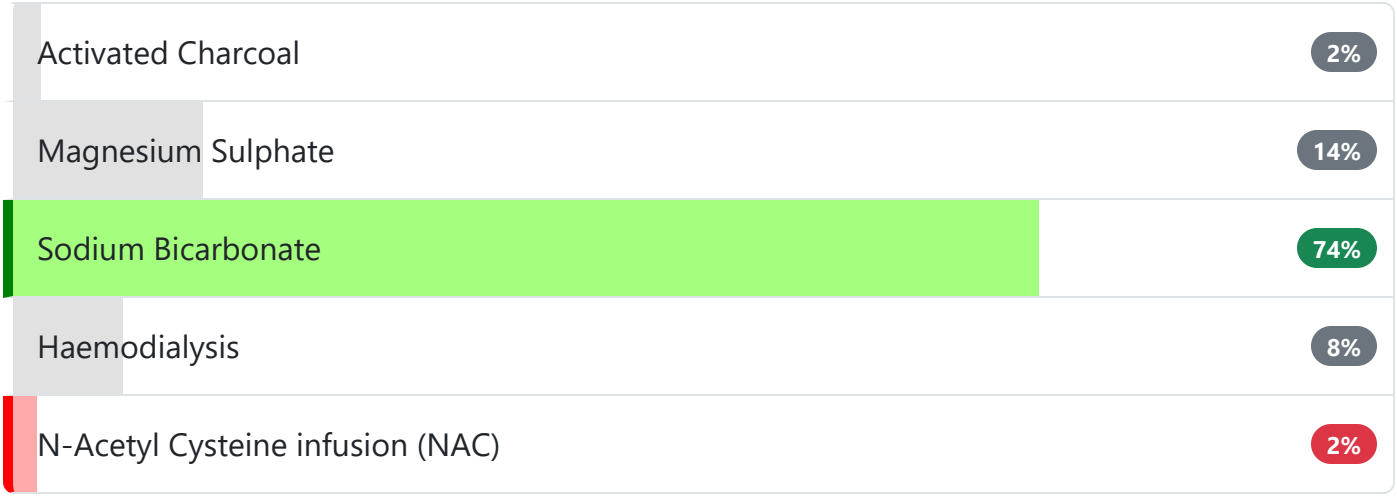
Na+	136 mmol/l
K+	4.6 mmol/l
Urea	10.9 mmol/l
Creatinine	110 µmol/l
Serum bilirubin	30 µmol/l
Serum alkaline phosphatase	135 IU/l
Serum aspartate aminotransferase	50 IU/l
C Reactive protein	2mg/l
Haemoglobin	14.6 g/dl
White cell count	5.6 x 109/L
INR	1.4

ABG (on air):

pH	7.258
pO2	11.7 kPa
pCO2	3.4 kPa
Lactate	1.6 mmol/l
Base Excess	-8.4 mmol/l
Bicarbonate	11.9 mmol/l

ECG showed bradycardia with widened QRS complexes and giant inverted T waves.

After fluid resuscitation, what is the next single most important management step?



This patient has overdosed on a number of medications, the most toxic being codeine and flecainide. He has evidence of cardiac arrhythmias, bradycardia and metabolic acidosis consistent with flecainide overdose, and also mildly deranged liver functions. There is no evidence of respiratory depression.

Flecainide is a class 1c antiarrhythmic, which blocks sodium channels leaving the action potential unchanged. Cardiac features predominate, causing bradycardia, hypotension, nodal and ventricular tachycardia. ECG abnormalities include marked prolongation of the QRS and Q-T interval and giant inverted T waves. Other features include hypoxia, respiratory depression, metabolic acidosis, and reduced renal function. Nausea, vomiting, coma and convulsions have been reported.

As the time of ingestion is unknown, possibly up to 4 hours ago, he has probably missed the window for gastric decontamination with activated charcoal. The presence of QRS prolongation and acidosis would require rapid correction with sodium bicarbonate infusions, ideally via a central line, to reverse the effects of flecainide.

Haemodialysis are of no value in eliminating flecainide from the system. Magnesium is indicated if there is evidence of a prolonged QT interval. NAC is indicated for paracetamol overdose.

Valerian root tablets are herbal sleeping remedies and do not have lasting side effects if taken in large quantities.

👍 👎 💬 Discuss (4) Improve

Next question

Flecainide

Flecainide is a Vaughan Williams class 1c antiarrhythmic. It slows conduction of the action potential by acting as a potent sodium channel blocker (specifically the Nav1.5 sodium channels). This may be reflected by widening of the QRS complex and prolongation of the PR interval.

The Cardiac Arrhythmia Suppression Trial (CAST, 1989) investigated the use of agents to treat asymptomatic or mildly symptomatic premature ventricular complexes (PVCs) post myocardial infarction. The hypothesis was that this would reduce deaths from ventricular arrhythmias. Flecainide was actually shown to increase mortality post-myocardial infarction and is, therefore, contraindicated in this situation.

Indications

- atrial fibrillation
- SVT associated with accessory pathway e.g. Wolf-Parkinson-White syndrome

Contraindications

- post myocardial infarction
- structural heart disease: e.g. heart failure
- sinus node dysfunction; second-degree or greater AV block
- atrial flutter

Adverse effects

- negatively inotropic
- bradycardia
- proarrhythmic
- oral paraesthesia
- visual disturbances

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

1 2

Flecainide

Suggest link

Report broken link

Score: **18.1%**

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Back to top

A 59- year- old male presented with palpitations, excessive sweating and weight loss for two months. Two years ago he was diagnosed with atrial fibrillation and commenced on amiodarone.

On examination, his pulse rate is 80 beats per minute, irregularly irregular, and his blood pressure is 135/80. There was no goitre, no eye signs or hand signs.

Investigations reveal:

Serum free T4	60 pmol/l
Serum free T3	15 pmol/l (5-10)
Serum TSH	<0.05 mU/l
Serum antithyroid peroxidase	negative
TSH receptor antibodies	negative

Radioactive iodine uptake scan showed reduced uptake by the thyroid gland.

What is the most appropriate management for this patient?

- Discontinue amiodarone and give carbimazole
- Discontinue amiodarone and give potassium perchlorate
- Discontinue amiodarone and give prednisolone
- Radioactive iodine
- Total thyroidectomy

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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Investigations reveal:

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Serum free T3	15 pmol/l (5-10)
Serum TSH	<0.05 mU/l
Serum antithyroid peroxidase	negative
TSH receptor antibodies	negative

Radioactive iodine uptake scan showed reduced uptake by the thyroid gland.

What is the most appropriate management for this patient?

Discontinue amiodarone and give carbimazole	15%
Discontinue amiodarone and give potassium perchlorate	4%
Discontinue amiodarone and give prednisolone	78%
Radioactive iodine	2%
Total thyroidectomy	1%

The diagnosis is amiodarone induced thyrotoxicosis (AIT) type 2 which is a destructive thyroiditis treated with prednisolone.

AIT type 2 is differentiated from AIT type 1 by the reduced iodine uptake, absence of thyroid autoantibodies and absence of goitre.

AIT type 1 is treated with carbimazole or potassium perchlorate.

If amiodarone could not be stopped as in ventricular tachycardia, then total thyroidectomy should be considered.

Discuss (2) Improve

Next question

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link

Report broken link

Score: **18.1%**

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Back to top

An 85-year-old female is admitted under the general medical unit with acute thoracic back pain from a T6 crush fracture following a fall. She has a past history of systolic heart failure, depression and osteoporosis.

Her regular medications included aspirin, frusemide, spironolactone, bisoprolol, sertraline and calcium, vitamin D and weekly alendronate. These are continued throughout her admission.

Two days into her admission, the nurses note that she is agitated and a bit confused.

On examination, she looks flushed and is tachycardic with a heart rate of 120 beats/min and is hypertensive with a blood pressure of 185/70 mmHg, but is afebrile. Both her pupils are mildly dilated, she is mildly tremulous and is noted to have deep tendon hyperreflexia with easily inducible clonus.

Use of which of the following analgaesic medication could explain her current symptoms?

- Paracetamol
- Ibuprofen
- Oxycodone
- Tramadol
- Hydromorphone

Submit answer

Reference ranges

Score: **15.1%**

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Two days into her admission, the nurses note that she is agitated and a bit confused.

On examination, she looks flushed and is tachycardic with a heart rate of 120 beats/min and is hypertensive with a blood pressure of 185/70 mmHg, but is afebrile. Both her pupils are mildly dilated, she is mildly tremulous and is noted to have deep tendon hyperreflexia with easily inducible clonus.

Use of which of the following analgaesic medication could explain her current symptoms?

Paracetamol	1%
Ibuprofen	6%
Oxycodone	10%
Tramadol	**75%**
Hydromorphone	8%

Serotonin syndrome is a disorder characterised by serotonin excess, usually due to the use of 2 or more serotonergic drugs. Manifestations of the syndrome include changes in mental status, neuromuscular changes and autonomic overactivity. Clinically, this can be observed as hypertension, tachycardia, flushing and sweating, hyperflexia, clonus and muscle rigidity. Other potential signs include fever and changes in mental status, including agitation.

Serotonergic drugs that are associated with serotonin syndrome include tramadol, selective serotonin reuptake inhibitors (SSRI), monoamine oxidase inhibitors (MAOI), triptans and St Johns wort.

The management of serotonin syndrome involves discontinuation of all serotonergic drugs and

supportive care. If required, benzodiazepine can be administered to control agitation. In moderate to severe cases, 5-HT antagonists (e.g. cyproheptadine and chlorpromazine) are sometimes administered.

References:

- Hall M, Buckley N. Serotonin Syndrome. Aust Prescr. 2003;26:62-3
- Boyer EW, Shannon M. The serotonin syndrome. N Engl J Med. 2005 Mar 17;352(11):1112-20.

Discuss (1) | Improve

Next question

Serotonin syndrome

Causes

- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

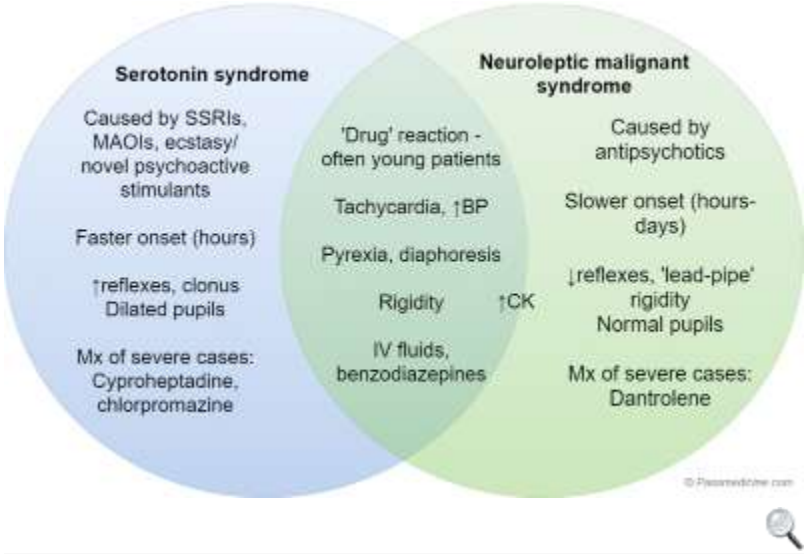
Features

- neuromuscular excitation
  - hyperreflexia
  - myoclonus
  - rigidity
- autonomic nervous system excitation
  - hyperthermia
  - sweating
- altered mental state
  - confusion

Management

- supportive including IV fluids
- benzodiazepines

- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

AI + Q 123

Next question

B *I* A

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe 1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

Report broken media

Score: **18.1%**

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Back to top

A 63 year old farmer is brought to the Emergency Department after falling into a trough of organophosphate sheep dip. On arrival in the department he is in extremis with profuse vomiting and productive of copious respiratory secretions. His airway is threatened but he is also agitated and difficult to assess. Respiratory rate is 20 breaths per minute and oxygen saturations are 87% on 4L/min oxygen. Wheeze is audible from the end of the bed. The heart rate is 55bpm and the blood pressure is 92/44mmHg. Heart sounds are normal. He is incontinent of urine and faeces and has severe abdominal pain. There is widespread muscle fasciculation and global weakness. Pupils are pinpoint.

Which of the following best describes the toxic action of organophosphate compounds?

- Adrenergic blockade due to competitive inhibition of α1, α2, β1 and β3 receptors ×
- Adrenergic upregulation due to inhibition of monoamine oxidase ×
- Cholinergic upregulation due to inhibition of acetylcholinesterase ×
- Cholinergic upregulation due to direct activation at muscarinic M1, M2 and M3 receptors ×
- Cholinergic upregulation due to direct activation at nicotinic CNS and muscle receptors ×

Submit answer

Reference ranges

Score: **15.1%**

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Which of the following best describes the toxic action of organophosphate compounds?

Adrenergic blockade due to competitive inhibition of α1, α2, β1 and β3 receptors	4%
Adrenergic upregulation due to inhibition of monoamine oxidase	1%
Cholinergic upregulation due to inhibition of acetylcholinesterase	65%
Cholinergic upregulation due to direct activation at muscarinic M1, M2 and M3 receptors	21%
Cholinergic upregulation due to direct activation at nicotinic CNS and muscle receptors	9%

Organophosphate poisoning occurs due to inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission

Important for me Less important

Organophosphate insecticides are highly effective pesticidal agents. They work by binding to and irreversibly inactivating the enzyme acetylcholinesterase (AChE) which is essential for respiration in insects. Unfortunately, they also inhibit AChE in humans too, which exerts toxic effects when inadvertent exposure occurs occupationally, and this class of agents can also be used as a chemical weapon in the form of nerve gases.

Due to the non-specificity of acetylcholinesterase in cholinergic synapses, inhibition of the enzyme leads to upregulation of neurotransmission at all cholinergic synapses, giving rise to both nicotinic and muscarinic symptoms. Classically, the muscarinic symptoms seen can be remembered by the mnemonic SLUDGE: salivation, lacrimation, urination, defecation, gastrointestinal pain, emesis, and also pupillary constriction, bronchoconstriction and bradycardia. Nicotinic symptoms include headache, anxiety, fasciculations, weakness, ataxia, confusion and respiratory depression.

Sometimes there is variation in the extent of symptoms observed due to the relative upregulation of each neurotransmitter symptom with either predominant nicotinic symptoms or muscarinic ones, for example either bradycardia due to muscarinic overdrive or tachycardia due to nicotinic drive.

Knowledge of the pathophysiological effects of organophosphates is important because it guides treatment. Severe exposure to organophosphates may cause death within minutes due to respiratory failure. Provision of supplemental oxygen is important. The first life-saving intervention which may be provided is intravenous atropine which is a competitive antagonist at muscarinic receptors, predominantly subtypes M1, M2 and M3. This increases heart rate and improves respiratory function by relaxing bronchial smooth muscle and drying secretions. Atropine administration may need to be repeated multiple times at doses up to 2mg every five minutes until heart rate is above 80. Definitive treatment may be provided by administration of pralidoxime or obidoxime which are oxime drugs which bind to an allosteric moiety of AChE and causes a conformational change in the enzyme allowing it to be released from the organophosphate. Organophosphates irreversibly bind to AChE when in their physiological state but conformational change by oximes allows this to be undone. This is time dependent however and a process known as aging occurs whereby the longer the enzyme is inhibited the less likely the oxime is to be able to reanimate it. In warfare, soldiers are often supplied with auto-injectors containing atropine, pralidoxime and midazolam as an immediate response to exposure to nerve agents.

While exposure to organophosphates often produces hypotension and bradycardia with bronchoconstriction, they have no effect on adrenergic neurotransmission.

It is also important to note that organophosphate exposure is from environmental sources and as such decontamination is vital to prevent exposure to healthcare providers and appropriate personal protective equipment must be worn by rescuers.

👍 👎 💬 Discuss (7) Improve

Next question ›

Organophosphate insecticide poisoning ★

One of the effects of organophosphate poisoning is inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission. In warfare, sarin gas is a highly toxic synthetic organophosphorus compound that has similar effects.

Features can be predicted by the accumulation of acetylcholine (mnemonic = SLUD)

- Salivation
- Lacrimation

- Urination
- Defecation/diarrhoea
- cardiovascular: hypotension, bradycardia
- also: small pupils, muscle fasciculation

Management

- atropine
- the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 49-year-old woman with a diagnosis of Hashimoto's thyroiditis is seen in clinic with recurrence of lethargy, constipation, cold intolerance and pedal oedema. These symptoms had previously resolved on starting treatment with levothyroxine.

Her co-morbidities include type 2 diabetes mellitus and hypertension. She was also diagnosed with mycobacterium tuberculosis of the lung five months ago. Her current medications are levothyroxine, amlodipine, ramipril, metformin, gliclazide, rifampicin, isoniazid and pyridoxine.

Her temperature is 36.5°C, pulse 55 beats per minute, blood pressure 165/102 mmHg and respiratory rate 15 breaths per minute.

Thyroid function tests showed:

Free thyroxine (T4)	5 pmol/L (10-25)
Free triiodothyronine (T3)	3 pmol/L (5-10)
Thyroid-stimulating hormone	7.2 mU/L (0.4-5.0)

What is the likely cause of her symptoms?

- Vitamin B6 deficiency
- Rifampicin
- Isoniazid
- Non-compliance with medications
- Amlodipine

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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Thyroid-stimulating hormone	7.2 mU/L (0.4-5.0)

What is the likely cause of her symptoms?

Vitamin B6 deficiency	4%
Rifampicin	64%
Isoniazid	15%
Non-compliance with medications	14%
Amlodipine	2%

Rifampicin is a P450 enzyme inductor

Important for me Less important

In patients with treated hypothyroidism, thyroxine requirements are increased by cytochrome P450 inducing drugs such as rifampicin. Isoniazid does not have this effect.

Non-compliance with levothyroxine may also give a similar picture, but there is no firm evidence

to suggest that this is the case, especially as the patient's symptoms had previously resolved.

Amlodipine can cause peripheral oedema, but it would not account for the other symptoms and investigation results.

Pyridoxine is a form of vitamin B6. Isoniazid interferes competitively with pyridoxine metabolism. However, a deficiency would typically present with a peripheral neuropathy.

Discuss (9) | Improve

Next question >

P450 enzyme system ★

Induction usually requires prolonged exposure to the inducing drug, as opposed to P450 inhibitors, where effects are often seen rapidly

Inducers of the P450 system include

- antiepileptics: phenytoin, carbamazepine
- barbiturates: phenobarbitone
- rifampicin
- St John's Wort
- chronic alcohol intake
- griseofulvin
- smoking (affects CYP1A2, reason why smokers require more aminophylline)

Inhibitors of the P450 system include

- antibiotics: ciprofloxacin, erythromycin
- isoniazid
- cimetidine,omeprazole
- amiodarone
- allopurinol
- imidazoles: ketoconazole, fluconazole
- SSRIs: fluoxetine, sertraline
- ritonavir
- sodium valproate
- acute alcohol intake
- quinupristin

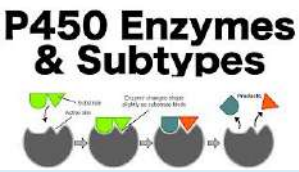
Next question

Textbooks

High-yield textbook

Extended textbook

Media



P450 Enzyme System (Inducers, Inhibitors, & Subtypes)

Dirty Medicine - YouTube 1 0

Report broken media

Score: **18.1%**

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A 52-year-old farmer was brought into the Emergency Department by his wife. Over the last few hours, he developed severe diarrhoea, passing 10 loose stools in the last four hours. He was also passing urine more frequently and on a couple of occasions was incontinent of urine. He also complained of feeling unwell with a headache and nausea and as well as a cough with white phlegm, and at times felt very short of breath particularly upon coughing. He denied the presence of chest pain. He had a past medical history of COPD, atrial fibrillation, depression and hypertension and his drug history comprised of Seretide 2puffs BD, salbutamol PRN, digoxin 250mcg OD, warfarin 2mg OD, sertraline 150mg OD, diazepam 2mg BD PRN and amlodipine 5mg OD.

Examination revealed the presence of an unwell and unkempt gentleman. His heart rate was 46 bpm and regular, his blood pressure was 88/48mmHG, his respiratory rate was 18, oxygen saturations 96% on air and temperature 35.7ºC. His BM was 3.8 mmol/l. Examination of his cardiovascular system revealed the presence of warm well perfused peripheries with normal heart sounds and a JVP of 3cm. Examination of his respiratory system revealed the presence of copious upper airways secretions but the absence of respiratory distress. Examination of his gastrointestinal system revealed the presence of excess salivation but was otherwise unremarkable. Examination of his neurological system revealed the presence of bilateral pupil constriction but otherwise no abnormalities and a GCS of 15.

He was promptly transferred to the resuscitation area and cannulated with 2 large bore cannulae. Stat intravenous saline 3 litres were promptly infused and an urgent medical consult was requested. Initial investigations revealed the following:

Hb	140 g/l
Platelets	198 * 10^9/l
WBC	6.6 * 10^9/l
CRP	4 mg/l

Coagulation screen: INR 2.2, APTT and fibrinogen within normal limits

Portable chest x-ray: poor quality rotated film. Normal appearance of heart and lung fields.

ECG: sinus bradycardia heart rate 45bpm, normal sinus rhythm, normal QRS and QTc intervals, no acute ST/T changes

Arterial blood gases on air:

pH	7.33
Pa02	12.6 KPa
PaCO2	5.8 kPa
HCO3	18 mmol/l
BE	-4

Of the following options which is the most appropriate curative management step?

- Commence digoxin fragment binding antibody (Digibind) ×
- Commence broad spectrum intravenous antibiotics and instigate sepsis protocol ×
- Commence intravenous noradrenaline ×
- Commence intravenous flumazenil ×
- Commence intravenous atropine ×

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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pH	7.33
Pa02	12.6 KPa
PaCO2	5.8 kPa
HCO3	18 mmol/l
BE	-4

Of the following options which is the most appropriate curative management step?

- Commence digoxin fragment binding antibody (Digibind) — 19%
- Commence broad spectrum intravenous antibiotics and instigate sepsis protocol — 2%
- Commence intravenous noradrenaline — 2%
- Commence intravenous flumazenil — 6%
- **Commence intravenous atropine** — 71%

Organophosphate insecticide - atropine

Important for me Less important

This gentleman has presented with organophosphate toxicity, as manifest by excess salivation, defecation, urinary frequency and copious respiratory secretions. His blood pressure was low and he was bradycardic. He had a history of depression and was prescribed a large dose of sertraline, and statistically was therefore at greater risk of deliberate self-harm (though it may also have been accidentally ingested). Of the following options, atropine is the only antidote to organophosphate and is, therefore, the most appropriate answer (note the question asked for the option most likely to lead to a cure, rather than the next best step, excluding option 3).

Of the remaining options, his symptoms do not correlate well with digoxin toxicity and his ECG has no changes other than bradycardia making this a less likely option. Even if he had ingested diazepam in excess there is no indication to commence flumazenil, and there is little evidence to suggest that he Is suffering from sepsis.

👍 👎 💬 Discuss (5) | Improve

Next question ›

Organophosphate insecticide poisoning

One of the effects of organophosphate poisoning is inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission. In warfare, sarin gas is a highly toxic synthetic organophosphorus compound that has similar effects.

Features can be predicted by the accumulation of acetylcholine (mnemonic = SLUD)

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Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 70-year-old man is admitted to hospital with shortness of breath and a productive cough. Following initial investigation a community-acquired pneumonia is diagnosed and treatment with IV fluids and IV antibiotics initiated promptly. Subcutaneous enoxaparin for venous thromboprophylaxis was initiated on the day of admission. Due to persistent hypotension and oliguria secondary due to presumed septic shock, the patient was admitted to the General Intensive Care Unit for inotropic support around 12 hours after initial presentation.

The patients past medical history featured a previous ST elevation myocardial infarction, treated with primary percutaneous coronary interventions one year previously. Regular medications included Aspirin, Clopidogrel, Ramipril, Bisoprolol and Atorvastatin. The patient had no known drug allergies and lived independently in a house with his wife.

With the above management, the patients condition stabilised and he was discharged to a respiratory ward on day 3 of the admission. Routine blood tests taken on day 7 of admission demonstrated a marked fall in platelet count compared to admission bloods. Full examination of the patient at this time did not demonstrate any evidence of venous thrombosis with no skin changes. Following advice from haematology further investigations were requested as detailed below.

Blood results	Day 0 (admission)	Day 3	Day 7
Platelets (x 10>3 / microlitre)	189	156	87
International normalised ratio	1.1	1.3	1.2

Heparin induced thrombocytopenia antibodies: positive (moderate titre)

What further investigation is required (if any) to confirm a diagnosis of heparin induced thrombocytopenia?

- Serotonin release assay
- Repeat heparin induced thrombocytopenia antibodies
- Venous doppler ultrasound of lower limbs
- Fibrinogen break-down products

No further investigation required

Submit answer

Reference ranges

Score: **15.1%**

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A 70-year-old man is admitted to hospital with shortness of breath and a productive cough. Following initial investigation a community-acquired pneumonia is diagnosed and treatment with IV fluids and IV antibiotics initiated promptly. Subcutaneous enoxaparin for venous thromboprophylaxis was initiated on the day of admission. Due to persistent hypotension and oliguria secondary due to presumed septic shock, the patient was admitted to the General Intensive Care Unit for inotropic support around 12 hours after initial presentation.

The patients past medical history featured a previous ST elevation myocardial infarction, treated with primary percutaneous coronary interventions one year previously. Regular medications included Aspirin, Clopidogrel, Ramipril, Bisoprolol and Atorvastatin. The patient had no known drug allergies and lived independently in a house with his wife.

With the above management, the patients condition stabilised and he was discharged to a respiratory ward on day 3 of the admission. Routine blood tests taken on day 7 of admission demonstrated a marked fall in platelet count compared to admission bloods. Full examination of the patient at this time did not demonstrate any evidence of venous thrombosis with no skin changes. Following advice from haematology further investigations were requested as detailed below.

Blood results	Day 0 (admission)	Day 3	Day 7
Platelets (x 10>3 / microlitre)	189	156	87
International normalised ratio	1.1	1.3	1.2

Heparin induced thrombocytopenia antibodies: positive (moderate titre)

What further investigation is required (if any) to confirm a diagnosis of heparin induced thrombocytopenia?

Serotonin release assay	19%
Repeat heparin induced thrombocytopenia antibodies	8%
Venous doppler ultrasound of lower limbs	3%
Fibrinogen break-down products	12%

No further investigation required 58%

The diagnosis of heparin induced thrombocytopenia is based on the correlation of the clinical presentation and laboratory results. It is helpful to calculate a pre-test probability score using a tool such as the Warkentin probability scale to assist with interpretation of HIT antibody results. In this case the patient has an intermediate clinical probability of HIT based on the fall in platelet count > 50 % of baseline between day 5-10 of initiating heparin therapy. However, the presence of an alternative cause of thrombocytopenia (severe sepsis) reduces the probability of HIT.

The combination of moderate clinical probability and moderate HIT antibody titre indicate the need for additional testing. Functional assays such as the serotonin release assay have a high sensitivity and specificity for HIT (> 95 %) and so will confirm or exclude a diagnosis of HIT.

Given the clinical picture, it would be prudent in this case to hold further heparin anticoagulants and introduce a non-heparin anticoagulant such as Bivalirudin to reduce the risk of HIT related thrombosis while further testing is being conducted.

Linkins L-A. Heparin induced thrombocytopenia. BMJ 2014;349:7566.

Discuss (5) Improve

Next question >

Heparin ★

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	Low molecular weight heparin (LMWH)
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF

3 2

Heparin

British Society for Haematology

2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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A 62-year-old man is seen in the Emergency Department with a rash which has developed over the last 24 hours. He has had increasing malaise throughout the day. His past medical history includes hypertension and gout. He was started on allopurinol 3 weeks ago due to increasing frequency of his gout attacks. He also takes ramipril and amlodipine.

On examination he has an erythematous maculopapular rash over his whole body, worst on his trunk. He is tachycardic at 105 beats per minute with a blood pressure of 110/55 mmHg and a temperature of 38.1 ºC. He has some residual pain in his right great toe but no other focal signs or symptoms.

His blood tests are as follows:

Hb	130 g/l	Na^+	141 mmol/l
Platelets	550 * 10^9/l	K^+	4.7 mmol/l
WBC	12.5 * 10^9/l	Urea	6 mmol/l
Neuts	6 * 10^9/l	Creatinine	95 µmol/l
Lymphs	5 * 10^9/l	CRP	65 mg/l
Eosin	1.5 * 10^9/l		

What is the most likely diagnosis?

- Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome
- Stephens-Johnson syndrome
- Anaphylactoid reaction
- Toxic epidermal necrolysis
- Reactive arthritis

Submit answer

Reference ranges

Score: **15.1%**

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Lymphs	5 * 10^9/l	CRP	65 mg/l
Eosin	1.5 * 10^9/l		

What is the most likely diagnosis?

Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome	89%
Stephens-Johnson syndrome	6%
Anaphylactoid reaction	2%
Toxic epidermal necrolysis	3%
Reactive arthritis	0%

The presence of maculopapular rash, fever and raised lymphocytes and eosinophils points to a likely diagnosis of DRESS (drug reaction with eosinophilia and systemic symptoms) syndrome. This is a rare complication following treatment with chemotherapy agents, allopurinol or anti-epileptics.

Stevens-Johnson syndrome and toxic epidermal necrolysis are part of a spectrum of skin reaction and can also occur with allopurinol. However typically patients present with a target-like rash and progressive blistering and eventual skin detachment. Eyes and other mucosal surfaces are often involved. Anaphylactoid reactions are immediately occurring and present with angioedema, bronchospasm and hypotension. Reactive arthritis presents with a triad of arthritis, urethritis and eye involvement.

Reference: DermNetNZ

Discuss (6) Improve

Next question

DRESS syndrome

DRESS is an unexpected, severe reaction to medication. Several organs are commonly affected including the skin, liver, kidneys, lungs and heart. Not all of these organs may be affected at any one time.

Classically patients develop a morbilliform skin rash in 80% cases which often leads to an exfoliative dermatitis, high fever, and inflammation of one or more organs. Vesicles and bullae may be seen. Erythroderma can occur in 10% of cases, mucosal involvement in 25% and facial swelling in 30%. The reaction usually occurs 2-8 weeks after commencing the offending drug.

Patients may develop systematic symptoms which can include haematological abnormalities (raised and low white count, eosinophilia (in 30% this is > $2.0 * 10^9$ /L, thrombocytopaenia, anaemia, atypical lymphocytes), enlarged lymph nodes (75%), 10% develop kidney disease which is usually mild (interstitial nephritis is common, renal failure is rare), myocarditis, pericarditis, liver enlargement, hepatitis and rarely hepatic necrosis with liver failure (abnormal liver function tests are found in 70-90% in particularly raised transaminases), lung disease (pneumonitis, pleuritis, pneumonia), neurological involvement which may lead to meningitis and encephalitis, gastrointestinal symptoms, in severe cases, acute colitis and pancreatitis can occur, and endocrine abnormalities may include thyroiditis and diabetes.

Diagnosis can be very difficult and it may also be difficult to determine the exact drug causing the hypersensitivity as first exposure may have started 8 weeks prior. Common drugs causing DRESS include allopurinol, anti-epileptics, antibiotics, immunosuppresants, HIV treatment and NSAIDS. A careful drug history should be taken.

The diagnosis should be suspected when there is presence of the triad of extensive skin rash, high

fever, and organ involvement, supported by a finding of eosinophilia and abnormal liver function tests. RegiSCAR has proposed a diagnostic criteria and patients require at least 3 of the following:

- Hospitalisation
- Reaction suspected to be drug related
- Acute skin rash
- Fever about 38ºC
- Enlarged lymph nodes at two sites
- Involvement of at least one internal organ
- Blood count abnormalities such as low platelets, raised eosinophils or abnormal lymphocyte count.

Skin biopsy can help to confirm the diagnosis. This may show inflammatory infiltrate in particularly eosinophils, extravasated erythrocytes and oedema. Regular blood tests including FBC, clotting, liver and renal function, CK, viral screen, glucose and thyroid function tests should be taken. Investigations looking for complications should be undertaken including ECG, CXR, echocardiogram, and urinalysis.

DRESS syndrome treatment requires all medications that are a possible culprits to be stopped and supportive care started. Antihistamines, topical steroids and emollients can be used to help control the rash. Careful fluid balance is necessary and clinicians should be aware of the patients nutritional status. Skin should be regularly checked and secondary infections should be treated with antibiotics. Systemic steroids may be started in severe cases where exfoliative dermatitis / pneumonitis / hepatitis is present. Occasionally immunosuppressants, intravenous immunoglobulin and plasmapheresis may be started. Potential culprit drugs should not be restarted again. The mortality is around 8%.

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Media

DRESS Syndrome

Armando Hasudungan - YouTube 6 0

Report broken media

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A 38-year-old man presents to the emergency department following the ingestion of a staggered paracetamol overdose, after an argument with his partner. He appears well and N-acetylcysteine is administered.

Twenty-four hours later, he deteriorates clinically and appears confused. Further blood tests are taken, the results are pending, and the medical team is discussing the option of a liver transplant.

What single feature would justify this?

- Arterial pH <7.3
- Creatinine >300 µmol/l
- Grade III or IV encephalopathy
- Prothrombin time >100 seconds
- Staggered overdose requiring N-acetylcysteine

Submit answer

Reference ranges

Score: **15.1%**

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A 38-year-old man presents to the emergency department following the ingestion of a staggered paracetamol overdose, after an argument with his partner. He appears well and N-acetylcysteine is administered.

Twenty-four hours later, he deteriorates clinically and appears confused. Further blood tests are taken, the results are pending, and the medical team is discussing the option of a liver transplant.

What single feature would justify this?

Arterial pH <7.3	68%
Creatinine >300 µmol/l	4%
Grade III or IV encephalopathy	14%
Prothrombin time >100 seconds	12%
Staggered overdose requiring N-acetylcysteine	3%

Liver transplantation criteria in paracetamol overdose: pH < 7.3 more than 24 hours after ingestion

Important for me Less important

The correct answer is an **arterial pH <7.3**. The King's College Hospital criteria define two categories of patients with poor prognosis following paracetamol overdose. One cohort is those in whom arterial pH is less than 7.3, over 24 hours after ingestion. The other cohort is patients who have all three of the following: creatinine >300 µmol/l, prothrombin time >100 seconds and grade III/IV encephalopathy. Therefore, of the options above, the only option which, alone, would warrant consideration of transplantation is arterial pH <7.3.

Creatinine >300 µmol/l alone is therefore incorrect. This forms part of three elements, as described above, which must all be present.

For the same reason, **grade III or IV encephalopathy** alone is also therefore incorrect. It must be present alongside elevated creatinine and prothrombin time.

Prolonged prothrombin time is also incorrect for the reasons described above.

Whether the overdose was **staggered and/or required N-acetylcysteine** infusion does not form part of the criteria.

Discuss | Improve

Next question >

Paracetamol overdose: management

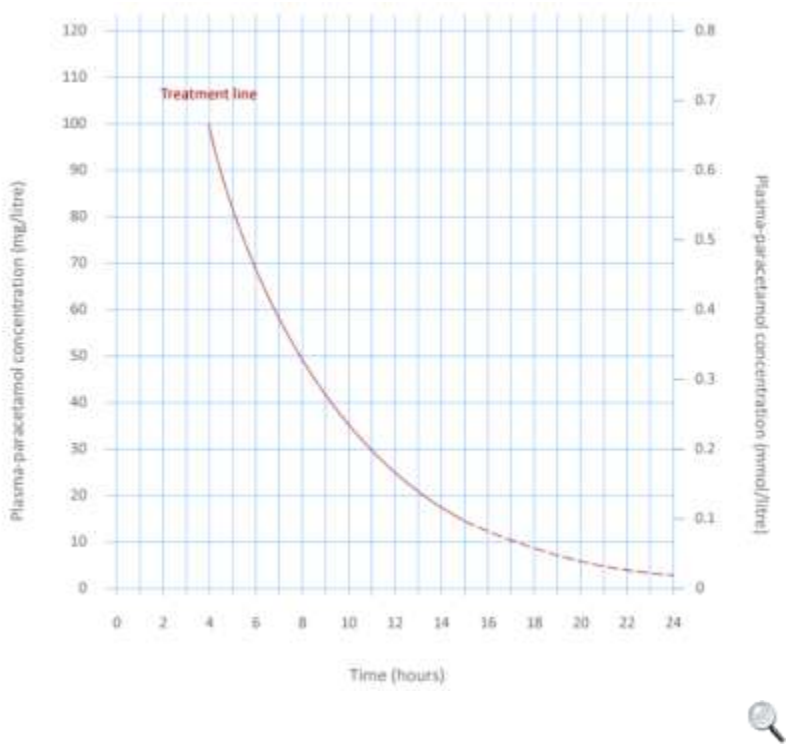
The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Medicines and Healthcare products Regulatory Agency

3 3

2012 Paracetamol overdose update

Suggest link Report broken link

Score: **18.1%**

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Back to top

A 19 year old man is brought into the Accident & Emergency department by his parents who are very concerned about his well being, and a medical opinion is sought. He arrived home from an evening with friends and was behaving bizzarely. When you talk to him, he tells you that he is feeling on top of the world and that he is an angel sent from god. He also describes seeing music as colours coming out of the radio earlier in the evening. He describes colours being more vivid and vibrant but denies any hallucinations. He has been unable to sleep for the past 24 hours

On examination, he is confused and disorientated but alert, skin colour is normal, central capillary refill time less than 2 seconds with moist mucous membranes. Normal eye movements, no peripheral focal neurology HR 115 sinus tachycardia (ECG normal) Blood tests (essentially normal) RR 16. Pupils dilated but responsive to light equally BP 174/98. When you are testing his eye movements he describes seeing trailing colours and when he closes his eyes he can see after-images that persist for a few seconds. He repeatedly asks you for glasses of water during the consultation and drinks them quite rapidly.

His mother says that he has no past history of medical problems and that his mental state has been normal until today and that he was well before he left the house 6 hours ago this evening. There is a family history of depression and he has recently left university due to not achieving the required grades

Towards the end of the consultation, he suddenly becomes very agitated and fearful, pointing at a wall believing that he is being chased by a monster, his family are unable to comfort him or lessen his agitation.

What is the the most appropriate treatment to give at this time?

- Haloperidol 5mg IM
- Lorazepam 1mg IM
- Lorazepam 1mg PO
- Olanzapine 10mg PO
- Lorazepam IV 1mg and cyproheptadine.

Submit answer

Reference ranges

Score: **15.1%**

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Towards the end of the consultation, he suddenly becomes very agitated and fearful, pointing at a wall believing that he is being chased by a monster, his family are unable to comfort him or lessen his agitation.

What is the the most appropriate treatment to give at this time?

Haloperidol 5mg IM	23%
Lorazepam 1mg IM	33%
Lorazepam 1mg PO	18%
Olanzapine 10mg PO	5%
Lorazepam IV 1mg and cyproheptadine.	21%

This man is suffering from LSD intoxication. He is describing the symptom of synaesthesia where sensations are perceived in a different modality, in this case seeing music He is also describing palinopsia which is where an after image persists for a few seconds after looking away from the original stimulus. This can happen in a clear sensorium if on focuses on an image for long enough but then the colours will be inverted. In palinopsia there is no inversion of colours. Palinopsia and synaesthesia can occur in temporal lobe epilepsy and other organic conditions, but is quite typical of hallucinogenic usage, particularly LSD. LSD intoxication can also cause expansive mood and grandiosity as seen here and this may rise to delusional intensity. Illusions (distortions of normal perception) and hallucinations in any modality are relatively common as is agitation and anxiety.

The answer is C. In the fist instance reassurance should be used and the patient should be placed in a calm environment with familiar people around. If this fails then benzodiazepenes should be administered. They should be tried orally first as the rate of action PO is similar to IM and injection may cause unecceasry distress in an agitated patient. The question tells you that he has drank water during the consultation, so it is reasonable to try PO first, if this fails then IM would be the next line.

Options A and D could be used as second line therapy if benzodizaepenes fail and also can be used in the treatment of mania. Mania is not the diagnosis here as there is a short history of onset, palinopsia and synaesethesia are much less common and you should not see dilated pupils.

Option E would be a treatment for severe seritonergic syndrome. LSD intoxication can cause serotinergic syndrome, especially if taken in conjunction with other drugs such as MDMA, SSRIs or TCAS. However, there is no evidence of this in our individual.

Discuss (11) | Improve

Next question >

LSD intoxication

Lysergic acid diethylamide (LSD) is the first synthetic hallucinogen which gained popularity in the 1960s to 1980s as a recreational drug. Its psychedelic effects usually involve heightening or distortion of sensory stimuli and enhancement of feelings and introspection. It is one of the most potent psychoactive compounds known.

Epidemiology

- Its usage has declined in recent years but still persists.

- The Drug Abuse Warning Network (DAWN) estimated that in 2011, the incidence of LSD-related emergency department attendance in the U.S. is 1.5 per 100,000 population.
- Adolescents and young adults are the most frequent users.

Patients with LSD toxicity typically present following acute panic reactions (known as bad trips), massive ingestions or unintentional ingestions.

Psychoactive symptoms

- Variable subjective experiences
- Impaired judgements which can lead to injury
- Amplification of current mood which leads to euphoria or dysphoria
- Agitation, appearing withdrawn - especially in inexperienced users
- Drug-induced psychosis

Somatic symptoms

- Nausea
- Headache
- Palpitations
- Dry mouth
- Drowsiness
- Tremors

Signs

- Tachycardia
- Hypertension
- Mydriasis
- Paresthesia
- Hyperreflexia
- Pyrexia

Manifestations such as tachycardia, hypertension, pupillary dilation, tremor, and hyperpyrexia can occur within minutes following oral administration of 0.5-2 µg/kg.

Massive overdoses can lead to the following complications:

- Respiratory arrest
- Coma
- Hyperthermia
- Autonomic dysfunction
- Bleeding disorders

Investigations:

- The diagnosis of LSD toxicity is mainly based on history and examination.
- Most urine drug screens do not pick up LSD.

Management

- Management of the intoxicated patient is dependent on the specific behavioural manifestation elicited by the drug.
- Agitation, e.g. from a 'bad trip', should be first managed with supportive reassurance in a calm, stress-free environment. If ineffective, benzodiazepines are the medication of choice.
- LSD-induced psychosis may require antipsychotics.
- Massive ingestions of LSD should be treated with supportive care, including respiratory support and endotracheal intubation if needed. Hypertension, tachycardia, and hyperthermia should be treated symptomatically. Hypotension should be treated initially with fluids and subsequently with vasopressors if required.
- Because LSD is rapidly absorbed through the gastrointestinal tract, activated charcoal administration and gastric emptying are of little clinical value by the time a patient presents to the emergency department.

AI + Q 123

Next question ›

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 66-year-old woman is reviewed in the rheumatology clinic for ongoing rheumatoid arthritis, which has been difficult to control. Several joints are currently flaring up and her current agent is not effective in controlling her symptoms.

After some consideration, she is switched to another agent. Blood tests are measured before she starts:

eGFR	>90 ml/min/1.73 m^2	(>90)
Creatinine	56 µmol/L	(55 - 120)

Several weeks after commencing the new drug, her blood tests are measured again:

eGFR	27 ml/min/1.73 m^2	(>90)
Creatinine	198 µmol/L	(55 - 120)

What is she most likely to have started?

- Ciclosporin
- Etanercept
- Infliximab
- Leflunomide
- Sulfasalazine

Submit answer

Reference ranges

Score: **15.1%**

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eGFR	27 ml/min/1.73 m^2	(>90)
Creatinine	198 µmol/L	(55 - 120)

What is she most likely to have started?

Ciclosporin	68%
Etanercept	5%
Infliximab	5%
Leflunomide	12%
Sulfasalazine	10%

Ciclosporin may cause nephrotoxicity

Important for me Less important

Of the options above, **ciclosporin** is best known to cause nephrotoxicity. It is sometimes used as a disease-modifying agent for rheumatoid arthritis. As well as being nephrotoxic, it is hepatotoxic, causes fluid retention, hyperkalaemia, hypertrichosis, gingival hyperplasia and other side effects.

Etanercept is a newer, biologic drug that interferes with tumour necrosis factor-alpha. It is not

widely known to be nephrotoxic. There are some case reports but overall ciclosporin is a much more likely cause.

Infliximab is another biologic used in the management of rheumatoid arthritis and also interferes with tumour necrosis factor-alpha. Like etanercept, it is not particularly known for nephrotoxicity, unlike cyclosporin.

Leflunomide has a similar side effect profile to methotrexate, affecting a wide number of systems, but is not strongly associated with nephrotoxicity.

Sulfasalazine is an aminosalicylate, sometimes used in the management of rheumatoid arthritis. It is not strongly linked to nephrotoxicity.

Discuss | Improve

Next question >

Ciclosporin ★

Ciclosporin is an immunosuppressant which decreases clonal proliferation of T cells by reducing IL-2 release. It acts by binding to cyclophilin forming a complex which inhibits calcineurin, a phosphatase that activates various transcription factors in T cells

Adverse effects of ciclosporin (note how everything is increased - fluid, BP, K^+, hair, gums, glucose)

- nephrotoxicity
- hepatotoxicity
- fluid retention
- hypertension
- hyperkalaemia
- hypertrichosis
- gingival hyperplasia
- tremor
- impaired glucose tolerance
- hyperlipidaemia
- increased susceptibility to severe infection

Interestingly for an immunosuppressant, ciclosporin is noted by the BNF to be 'virtually non-myelotoxic'.

Notable interactions

- cannabidiol: may increase the concentration of ciclosporin BNF

Indications

- following organ transplantation
- rheumatoid arthritis
- psoriasis (has a direct effect on keratinocytes as well as modulating T cell function)
- ulcerative colitis
- pure red cell aplasia

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 2 1

Ciclosporin

British Association of Dermatologists 1 1

2018 Guidelines for the safe and effective prescribing of oral ciclosporin in dermatolog

Suggest link Report broken link

Score: **18.1%**

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Investigations:

Hb	11.3 g/dl
MCV	102 fl
Platelets	110 * 10^9/l
WBC	12 * 10^9/l
Creatinine	117 umol/L
Urea	9 umol/L
Na+	146 mmol/L
K+	5.4 mmol/L
CL-	109 mmol/L

Arterial blood gas:

pH	7.21
PaO2	10.9 kPa
PaCO2	4.2 kPa
HCO3	16 mEq/L

Considering the ingested toxin, what is the next best step in your management?

- Fomepizole
- N-acetyl cysteine
- Activated charcoal

- Gastric lavage
- Sodium bicarbonate

Submit answer

Reference ranges

Score: **15.1%**

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Creatinine	117 umol/L
Urea	9 umol/L
Na+	146 mmol/L
K+	5.4 mmol/L
CL-	109 mmol/L

Arterial blood gas:

pH	7.21
PaO2	10.9 kPa
PaCO2	4.2 kPa
HCO3	16 mEq/L

Considering the ingested toxin, what is the next best step in your management?

Fomepizole	90%
N-acetyl cysteine	1%
Activated charcoal	2%

Gastric lavage	1%
Sodium bicarbonate	7%

Answer: Fomepizole or 4-methylpyrazole.

This patient has ingested methanol. Clue to this is the fact that he smells of alcohol but denies having consumed any, though definitive diagnosis is based on a raised serum methanol level. Initial symptoms are similar to those felt in alcohol intoxication including ataxia, disinhibition, headaches, nausea and vomiting. These are followed by drowsiness which may progress to coma. Metabolic acidosis with raised anion gap is common.

He has visual impairment indicating severe methanol toxicity. Visual signs present in methanol toxicity include, photophobia, blurred vision, poor visual acuity and blindness. Fundoscopy could reveal optic disc swelling or pseudoglaucomatous changes.

This patient needs urgent treatment with i.v fomepizole. If this is not available then i.v ethanol is an adequate alternative though it will produce symptoms similar to alcohol intoxication.

Fomepizole acts by inhibiting the primary step in the conversion of methanol to formic acid, a highly toxic metabolite. Specifically it competitively inhibits alcohol dehydrogenase, the enzyme that converts methanol to formaldehyde which is then further oxidised to formic acid by formaldehyde dehydrogenase.

The signs of methanol toxicity are caused my formic acid so inhibition of its synthesis is a very important step in its management.

Dialysis may be needed to help rapid elimination of the toxin from the system. The following are the indications for haemodialysis in methanol intoxication:

- worsening acidosis despite sodium bicarbonate treatment
- visual involvement
- quantity of consumed methanol exceeding 30ml
- serum methanol level greater than 20mg/dl

Other conditions that may be present in methanol toxicity include:

- seizures
- cardiac arrhythmias and heart failure
- haemorrhagic pancreatitis
- parkinsonism secondary to haemorrhagic and non haemorrhagic damage to the basal ganglia

He is acidotic and whilst sodium bicarbonate could help with acidosis it is unlikely to lead to

overall improvement.

N-acetyl cysteine is used in paracetamol overdose.

Further reading:
Acute bilateral blindness caused by accidental methanol intoxication during fire 'eating'
C Cursiefen, A Bergua http://bjo.bmj.com/content/86/9/1064.full

Discuss (8) Improve

Next question

Methanol poisoning

Methanol poisoning causes both the effects associated with alcohol (intoxication, nausea etc) and also specific visual problems, including blindness. These effects are thought to be secondary to the accumulation of formic acid. The actual pathophysiology of methanol-associated visual loss is not fully understood but it is thought to be caused by a form of optic neuropathy

Management
- fomepizole (competitive inhibitor of alcohol dehydrogenase) or ethanol
- haemodialysis
- cofactor therapy with folinic acid to reduce ophthalmological complications

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

1 5

Suggest link

Report broken link

Score: **18.1%**

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Back to top

A 49-year-old woman comes to the Emergency department suffering from nausea and lethargy which has increased over the past few days. She has undergone a renal transplant for end stage renal failure due to chronic reflux nephropathy some 3 months ago. You understand her GP prescribed an antibiotic for a respiratory tract infection without checking for potential interactions with her ciclosporin based immunosuppressive regime. Creatinine has increased significantly, and ciclosporin is above the upper limit of the recommended range.

Investigations:

Na^+	142 mmol/l
K^+	5.1 mmol/l
Urea	8.2 mmol/l
Creatinine (3 months ago)	161 µmol/l
Creatinine (today)	225 µmol/l

Which of the following antibiotics is she most likely to have been prescribed?

- Amoxicillin
- Cephalexin
- Clarithromycin
- Doxycycline
- Levofloxacin

Submit answer

Reference ranges

Score: **15.1%**

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Na^+	142 mmol/l
K^+	5.1 mmol/l
Urea	8.2 mmol/l
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Creatinine (today)	225 µmol/l

Which of the following antibiotics is she most likely to have been prescribed?

Amoxicillin	2%
Cephalexin	3%
Clarithromycin	73%
Doxycycline	4%
Levofloxacin	19%

The key here is to recognise that ciclosporin is metabolised by CYP3A4, and clarithromycin and erythromycin are both potent CYP3A4 inhibitors, leading to a potential elevation in ciclosporin levels, and subsequent nephrotoxicity. Because of the danger of either CYP3A4 inhibition or activation in patients taking ciclosporin, patients are advised to consult their transplant team before starting any new medication.

Amoxicillin is predominantly excreted unchanged in the urine, as is cephalexin. Levofloxacin undergoes very limited biotransformation, with 85% excreted in the urine. Doxycycline is

concentrated in bile, not metabolised via the P450 system. After 3 days around 40% is excreted in the urine and approximately 30% in faeces.

Discuss (3) Improve

Next question

Ciclosporin

Ciclosporin is an immunosuppressant which decreases clonal proliferation of T cells by reducing IL-2 release. It acts by binding to cyclophilin forming a complex which inhibits calcineurin, a phosphatase that activates various transcription factors in T cells

Adverse effects of ciclosporin (note how everything is increased - fluid, BP, K^+, hair, gums, glucose)

- nephrotoxicity
- hepatotoxicity
- fluid retention
- hypertension
- hyperkalaemia
- hypertrichosis
- gingival hyperplasia
- tremor
- impaired glucose tolerance
- hyperlipidaemia
- increased susceptibility to severe infection

Interestingly for an immunosuppressant, ciclosporin is noted by the BNF to be 'virtually non-myelotoxic'.

Notable interactions

- cannabidiol: may increase the concentration of ciclosporin BNF

Indications

- following organ transplantation
- rheumatoid arthritis
- psoriasis (has a direct effect on keratinocytes as well as modulating T cell function)
- ulcerative colitis
- pure red cell aplasia

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

2 1

Ciclosporin

British Association of Dermatologists

1 1

2018 Guidelines for the safe and effective prescribing of oral ciclosporin in dermatolog

Suggest link

Report broken link

Score: **18.1%**

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Back to top

A 48-year-old woman is brought in by ambulance to the emergency department, having been found at home by her neighbour with a decreased level of consciousness. She has a past medical history of open-angle glaucoma and is on regular acetazolamide.

On examination, she is obtunded. Her observations are heart rate of 111 beats per minute, blood pressure 93/65mmHg, respiratory rate of 22/min, temperature 36.7°C, oxygen saturations of 94% on room air. Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable. Her Glasgow coma scale is 10/15 (E2, V3, M5).

IV fluids are administered and arterial blood gas and blood tests are obtained.

Arterial blood gas results:

pH	7.24	(7.35-7.45)
paO2	10.2 kPa	(10-14)
paCO2	5.5 kPa	(4.5 - 6.0
Base excess	-4.2 mmol/L	(-2 - +2)
Chloride	95 mmol/L	(95-105)
Bicarbonate	14 mmol/L	(22-26)
Glucose	5.0 mmol/L	(4-11)
Lactate	2.2 mmol/L	(0.5-2.0)
SaO2	95 %	(94-98)

Blood test results:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.4 * 10^9/L	(4.0 - 11.0)
Na^+	140 mmol/L	(135 - 145)
K^+	5.0 mmol/L	(3.5 - 5.0)
Urea	8.0 mmol/L	(2.0 - 7.0)

Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	320 mOsm/Kg	(280-290)

What is the most likely diagnosis?

- Acetazolamide administration
- Addisonian crisis
- Ethylene glycol administration
- IV sodium chloride 0.9% administration
- Type 1 renal tubular acidosis

Submit answer

Reference ranges

Score: **15.1%**

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Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	320 mOsm/Kg	(280-290)

What is the most likely diagnosis?

Acetazolamide administration	16%
Addisonian crisis	3%
Ethylene glycol administration	76%
IV sodium chloride 0.9% administration	1%
Type 1 renal tubular acidosis	4%

Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

Ethylene glycol administration is the correct answer. This is a case of a raised anion gap metabolic acidosis with a raised osmolar gap. The anion gap is calculated as (sodium + potassium) - (chloride + bicarbonate). In this case, it is 36 mmol/L, which is raised (normal range 8-16 mmol/L). The osmolar gap is calculated as osmolality (measured) - osmolarity (calculated). Osmolarity (calculated) is [2*sodium + glucose + urea]. In this case, it is (320) - (293) = 27mOsm (normal range < 10), indicating a raised osmolar gap. A high osmolar gap means the presence of an unmeasured solute in high amounts e.g. ethylene glycol in this case.

Acetazolamide administration is incorrect. This causes a normal anion gap metabolic acidosis.

Addisonian crisis is incorrect. This is a cause of a normal anion gap metabolic acidosis. The anion gap is raised in this case.

IV sodium chloride 0.9% administration is incorrect. This will cause a hyperchloremic acidosis with a normal anion gap.

Type 1 renal tubular acidosis is incorrect. This is a cause of a normal anion gap metabolic acidosis.

Discuss (7) Improve

Next question

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **18.1%**

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Back to top

A 62-year-old gentleman presents to the emergency department with central chest pain. He describes his pain as a heavy pressure in the middle of his chest which radiates to his left arm. It has improved with the use of his GTN spray, but he did not want to use more than two puffs as he is concerned about getting a headache with its us. He has a past medical history of angina, previous myocardial infarction three years ago and he is a current smoker with a 25 pack-year history.

His ECG demonstrates ST depression in V3 to V5 of 2mm, pathological Q waves and T wave inversion. His troponin is raised at 350ng/L. He is given aspirin, clopidogrel and morphine. His pain settles and he starts to feel comfortable. He has a high GRACE (Global Registry of Acute Coronary Events) score at 122, meaning that he has a 7.9% chance of death within 6 months. Following discussion with the cardiology registrar will be scheduled for an angiogram within 12 hours. How should his acute coronary syndrome be further managed?

- Fondaparinux
- No further anticoagulants
- Unfractionated heparin
- Heparin sodium
- Warfarin

Submit answer

Reference ranges

Score: **15.1%**

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Fondaparinux	50%
No further anticoagulants	10%
Unfractionated heparin	34%
Heparin sodium	5%
Warfarin	1%

The correct answer is unfractionated heparin. This is a patient who has presented with chest pain and diagnosed with an NSTEMI and deemed to be at sufficiently at high risk to warrant an urgent angiography. NICE recommends that patients should be treated with aspirin and clopidogrel immediately and this should be combined with fondaparinux unless at high risk of bleeding or angiography is planned within 24 hours. If angiography is planned, then unfractionated heparin should be used. This is because angiography has a risk of haematoma formation and this risk is increased when fondaparinux is used compared to unfractionated heparin as the latter has a much shorter half-life.

Source:

'Unstable angina and NSTEMI: early management' Clinical guideline [CG94]. The National Institute

for Health and Care Excellence, March 2010.

Discuss (12) Improve

Next question

Heparin

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)

	Standard heparin	Low molecular weight heparin (LMWH)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

AI + Q 123

Next question ›

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BNF 3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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Back to top

A 43-year-old man attends the emergency department complaining of visual blurring, headache and nausea. He has a past medical history of alcohol dependency. He is not on any regular medications. He smokes 20 cigarettes daily and drinks at least one bottle of vodka daily. He is homeless and occasionally sources alcohol from other members of the homeless community.

His observations are as follows: heart rate 111 beats per minute, respiratory rate 28/minute, oxygen saturations 97% on room air, blood pressure 123/77 mmHg and temperature 37.1ºC.

He appears intoxicated. The cardio-respiratory examination is normal. His abdomen is mildly tender but soft and bowel sounds are present. Neurological examination is difficult but there are appears to be some blurring of his vision. His pupils are dilated. There is no epithelial defect upon staining of the eye.

Venous blood gas:

pH	7.31	(7.35-7.45)
HCO3	15 mmol/L	(22-26)
lactate	2.8 mmol/L	(0-2)
Potassium	4 mmol/L	(3.5-5.5)
Sodium	138 mmol/L	(135-145)
Cl-	96 mEq/L	(98-106

Based on the likely diagnosis, what is the most appropriate medication regimen?

- Folinic acid
- IV acetazolamide
- Prednisolone
- Thrombolysis
- Topical antibiotics

Submit answer

Reference ranges

Score: **15.1%**

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Sodium	138 mmol/L	(135-145)
Cl-	96 mEq/L	(98-106

Based on the likely diagnosis, what is the most appropriate medication regimen?

Folinic acid	74%
IV acetazolamide	19%
Prednisolone	6%
Thrombolysis	1%
Topical antibiotics	0%

Folinic acid can be used in methanol poisoning to reduce ophthalmological complications

Important for me Less important

Folinic acid is the correct answer. This patient is likely to have methanol poisoning, based on the history of nausea, tachypnoea, visual disturbance, elicit alcohol use and raised anion gap (31) metabolic acidosis. Folinic acid can be used as adjunctive treatment in methanol poisoning to reduce the risk of ophthalmological complications. Anchor treatments for methanol poisoning include ethanol/fomepizole, sodium bicarbonate +/- haemodialysis if necessary.

Prednisolone is incorrect. This can reduce the risk of visual complications in giant cell arteritis but this man is too young to have a diagnosis of giant cell arteritis, despite his headache.

IV acetazolamide is incorrect. This can be used to reduce the risk of visual complications in acute angle-closure glaucoma. However, it typically causes a unilateral red eye, which is not mentioned here.

Topical antibiotics is the wrong answer. The absence of an epithelial defect in the eye makes a corneal ulcer and infection unlikely.

Thrombolysis is incorrect. The clinical history suggests methanol poisoning rather than a stroke as a cause of visual loss.

Discuss (4) Improve

Next question

Methanol poisoning

Methanol poisoning causes both the effects associated with alcohol (intoxication, nausea etc) and also specific visual problems, including blindness. These effects are thought to be secondary to the accumulation of formic acid. The actual pathophysiology of methanol-associated visual loss is not fully understood but it is thought to be caused by a form of optic neuropathy

Management

- fomepizole (competitive inhibitor of alcohol dehydrogenase) or ethanol
- haemodialysis
- cofactor therapy with folinic acid to reduce ophthalmological complications

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

1 5

Suggest link

Report broken link

Score: **18.1%**

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A 48-year-old female presented with general fatigue, and nausea. She denies having pyrexia, vomiting diarrhoea or any pains. She appeared dehydrated but otherwise, observations and physical examination were within normal limits.

Blood results are as follows:

Na^+	130 mmol/l
K^+	4.8 mmol/l
Urea	18 mmol/l
Creatinine	162 µmol/l
Lithium	1.6 mmol/l (0.4-1.0 mmol/l)

Looking back at past results from 3 weeks ago, her renal function was in the normal range.

She has a history of bipolar disorder, diet-controlled diabetes and hypertension. She has been compliant with her lithium tablets and undergoing regular checks for the levels.

The patient revealed that her General Practitioner had recently started her on a new tablet 2 weeks ago.

Which of the following would most likely be the precipitant for her symptoms?

- Indapamide
- Amlodipine
- Dihydrocodeine
- Spironolactone
- Propranolol

Submit answer

Reference ranges

Score: **15.1%**

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The patient revealed that her General Practitioner had recently started her on a new tablet 2 weeks ago.

Which of the following would most likely be the precipitant for her symptoms?

- Indapamide — 75%
- Amlodipine — 2%
- Dihydrocodeine — 5%
- Spironolactone — 12%
- Propranolol — 6%

Lithium toxicity can be precipitated by thiazides

Important for me Less important

Acute renal impairment and lithium toxicity can be precipitated by any medicines that can impair renal function or induce hyponatraemia:

- Angiotensin-converting enzyme (ACE) inhibitors
- Diuretics (particularly thiazides including bendroflumethiazide and indapamide)
- Non-steroidal anti-inflammatory drugs (NSAIDs)

Target concentrations of lithium:

- acute episode (mania, hypomania, depression) 0.6-1.0 mmol/L (elderly 0.4-0.8 mmol/L).
- prophylaxis of bipolar affective disorder 0.4-0.8 mmol/L.
- toxic range usually >1.5 mmol/L; however, may begin at >1.0 mmol/L (levels >2 mmol/L need urgent treatment).

Discuss (2) | Improve

Next question >

Lithium toxicity ★

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure
- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- hyperreflexia
- acute confusion
- polyuria
- seizure
- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this
  - by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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An elderly lady brings her 32-year-old lodger into the emergency department after she witnesses him having two generalised seizures, lasting for around 4 minutes before spontaneously terminating, followed by a loss of consciousness lasting for around 10 minutes before slowly becoming rousable again. He is not known to have a history of epilepsy and does not take any regular medications. He denies any history of illicit drugs, smoking or excessive alcohol intake, corroborated by his landlady, who reports not to have seen him ever drinking in the 4 years he has been living in her loft space. Apart from a mild headache, he claims to have been well recently, continuing to work on his start-up computer software in the loft. She states that he has always 'kept himself to himself' in a cluttered dark loft space without a window, with a simple mattress and a small wardrobe. He didn't even ask for an additional heater during the cold winter as he thought 'the loft gas boiler provides enough warmth'. On examination, he is slightly sleepy but rousable. His Glasgow Coma Score is E4 V5 M6. Apart from erythematous lips, you note no other remarkable features. Neurological examination of his cranial nerves, upper and lower limbs are unremarkable. There is no ophthalmoscope available. His observations are heart rate 90/min and regular, BP 132/88 mmHg, sats 98% on air.

His blood tests demonstrate:

Hb	159 g/l
Platelets	282 * 10^9/l
WBC	7.9 * 10^9/l

Na^+	143 mmol/l
K^+	4.2 mmol/l
Urea	6.1 mmol/l
Creatinine	88 µmol/l
CRP	3 mg/l

His arterial blood gas demonstrate:

pH 7.25	
PaO2	15.8 kPa
PaCO2	2.9 kPa
Lactate	4.5 mmol/l

HbCO	24%

A CT head demonstrates a small area of acute ischaemic change in the right globus pallidus.

Which is the most appropriate next management?

- ○ High flow oxygen ×
- ○ Aspirin 300mg ×
- ○ Clopidogrel 300mg ×
- ○ PO chlordiazepoxide 40mg QDS ×
- ○ Start levetiracetam 250mg BD ×

Submit answer

Reference ranges

Score: **15.1%**

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A CT head demonstrates a small area of acute ischaemic change in the right globus pallidus.

Which is the most appropriate next management?

High flow oxygen	80%
Aspirin 300mg	11%
Clopidogrel 300mg	2%
PO chlordiazepoxide 40mg QDS	4%
Start levetiracetam 250mg BD	2%

The young patient presents with new seizures of on a background of a mild headache, red lips and a significantly raised COHb. This is a history suggestive of carbon monoxide poisoning. The ischaemic change on CT head, particularly in the globus pallidus with haemorrhagic transformation, a classic neurological consequence of carbon monoxide poisoning. It should be noted that pulse oximetry and PaO2 are commonly normal in CO poisoning. Carbon monoxide reduces haemoglobin binding capacity and not the amount of O2 dissolved in blood. A metabolic acidosis is present in the blood gas secondary to lactic acidosis from systemic ischaemia. If fundoscopy could have been performed, a cherry-red spot in the macula is the classic finding.

The initial management of CO poisoning is high flow oxygen, which reduces the half-life of CO binding to haemoglobin from 300 to 90 minutes. It is recommended that patients with severe acidosis (pH<7.1), COHb >25%, continued loss of consciousness and severe end organ failure be transferred to a unit for hyperbaric oxygen therapy.

Discuss (6) Improve

Next question >

Carbon monoxide poisoning ★

Carbon monoxide has a high affinity for haemoglobin and myoglobin resulting in a left-shift of the oxygen dissociation curve and tissue hypoxia. There are approximately 50 per year deaths from accidental carbon monoxide poisoning in the UK.

Pathophysiology

- carbon monoxide binds readily to haemoglobin, forming carboxyhaemoglobin → reduced oxygen-carrying capacity
- in carbon monoxide poisoning the oxygen saturation of haemoglobin decreases leading to an early plateau in the oxygen dissociation curve

Questions may hint at badly maintained housing e.g. student houses.

Features of carbon monoxide toxicity

- headache: 90% of cases
- nausea and vomiting: 50%
- vertigo: 50%
- confusion: 30%
- subjective weakness: 20%
- severe toxicity: 'pink' skin and mucosae, hyperpyrexia, arrhythmias, extrapyramidal features, coma, death

Investigations

- pulse oximetry may be falsely high due to similarities between oxyhaemoglobin and carboxyhaemoglobin
- therefore a venous or arterial blood gas should be taken
- typical carboxyhaemoglobin levels
  - < 3% non-smokers
  - < 10% smokers
  - 10 - 30% symptomatic: headache, vomiting
  - > 30% severe toxicity
- an ECG is a useful supplementary investgation to look for cardiac ischaemia

Management

- patients with suspected carbon monoxide poisoning should be assessed in the emergency department
- 100% high-flow oxygen via a non-rebreather mask
  - from a physiological perspective, this decreases the half-life of carboxyhemoglobin (COHb)
  - should be administered as soon as possible, with treatment continuing for a minimum of six hours
  - target oxygen saturations are 100%
  - treatment is generally continued until all symptoms have resolved, rather than monitoring CO levels
- hyperbaric oxygen

- due to the small number of cases the evidence base is limited, but there is some evidence that long-term outcomes may be better than standard oxygen therapy for more severe cases
- therefore, discussion with a specialist should be considered for more severe cases (e.g. levels > 25%)
- in 2008, the Department of Health publication 'Recognising Carbon Monoxide Poisoning' also listed loss of consciousness at any point, neurological signs other than headache, myocardial ischaemia or arrhythmia and pregnancy as indications for hyperbaric oxygen

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Media

Carbon Monoxide Poisoning

Kloss and Bruce - YouTube 3 2

Report broken media

Score: **18.1%**

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Back to top

A 65-year-old man presents to the emergency department with dysuria and abdominal pain. He has a past medical history of chronic kidney disease stage IV. His medications include atorvastatin, amlodipine and ramipril.

On examination, there is mild suprapubic tenderness.

Urinalysis:

Nitrites	positive
Leucocytes	+++
Blood	+
Protein	+
Glucose	negative

Blood tests:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	137 mmol/L	(135 - 145)
K^+	4.2 mmol/L	(3.5 - 5.0)
Urea	22.1 mmol/L	(2.0 - 7.0)
Creatinine	278 µmol/L	(55 - 120)
EGFR	23 mL/min/1.73m2	(90-120)
CRP	48 mg/L	(< 5)

Given the likely diagnosis, which antibiotic would be unsuitable?

- Amoxicillin
- Cephalexin

- Ciprofloxacin
- Co-amoxiclav
- Nitrofurantoin

Submit answer

Reference ranges

Score: **15.1%**

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Urea	22.1 mmol/L	(2.0 - 7.0)
Creatinine	278 µmol/L	(55 - 120)
EGFR	23 mL/min/1.73m2	(90-120)
CRP	48 mg/L	(< 5)

Given the likely diagnosis, which antibiotic would be unsuitable?

Amoxicillin	3%
Cephalexin	5%

Ciprofloxacin	28%
Co-amoxiclav	5%
Nitrofurantoin	59%

Nitrofurantoin is best avoided in patients with CKD stage 3 or higher due to the significant risk of treatment failure and occurrence of side effects due to drug accumulation

Important for me Less important

Nitrofurantoin is correct. The patient has a urinary tract infection. Nitrofurantoin is a reasonable choice in some circumstances. However, this antibiotic should be avoided in patients with CKD stage 3 or higher due to the significant risk of treatment failure and occurrence of side effects due to drug accumulation.

Amoxicillin is incorrect. This may be a suitable choice for a urinary tract infection in some circumstances. Again, a dose reduction could be considered in severe renal impairment but it is not contraindicated.

Cephalexin is not the right answer. This is a potential antibiotic choice for a urinary tract infection. The BNF states the dose adjustment for renal impairment is a max dose of 1.5 g daily if eGFR 10-40 mL/minute/1.73 m2.

Ciprofloxacin is not the right answer. This would be a suitable choice for a urinary tract infection in some circumstances. The BNF states the dose adjustment in renal impairment is to give 250-500 mg every 12 hours if eGFR 30-60 mL/minute/1.73 m2 or every 24 hours if eGFR is less than 30 mL/minute/1.73 m2.

Co-amoxiclav is not the right answer. This is a possible antibiotic choice for a urinary tract infection. The BNF states the dose adjustment in renal failure if eGFR 10-30 mL/minute/1.73 m2 is one 250/125 strength tablet every 12 hours or one 500/125 strength tablet every 12 hours.

Discuss (4) Improve

Next question ›

Prescribing in patients with renal failure ★

Questions regarding which drugs to avoid in renal failure are common

Drugs to avoid in renal failure

- antibiotics: tetracycline, nitrofurantoin
- NSAIDs
- lithium
- metformin

Drugs likely to accumulate in chronic kidney disease - need dose adjustment

- most antibiotics including penicillins, cephalosporins, vancomycin, gentamicin, streptomycin
- digoxin, atenolol
- methotrexate
- sulphonylureas
- furosemide
- opioids

Drugs relatively safe - can sometimes use normal dose depending on the degree of chronic kidney disease

- antibiotics: erythromycin, rifampicin
- diazepam
- warfarin

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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On examination, both upper and lower limbs are rigid and mildly bradykinetic. A bilateral resting tremor is noted in both hands. Reflexes and sensation are normal. Examination of cranial nerves and eye movements are unremarkable.

His initial blood tests return and are as follows:

Hb	163 g/l
Platelets	276 * 10^9/l
WBC	5.5 * 10^9/l
Vitamin B12	202 ng/l
Folate	471 (>317 nmol/l)

Na^+	142 mmol/l
K^+	4.3 mmol/l
Urea	6.4 mmol/l
Creatinine	85 µmol/l
CRP	7mg/l
HIV	negative

Which investigation is most likely to yield the underlying diagnosis?

- DAT scan
- Anti-GM1b antibodies
- Borrelia burgdorferi serology

- Urine porphybilinogen
- Serum heavy metals

Submit answer

Reference ranges

Score: **15.1%**

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CRP	7mg/l
HIV	negative

Which investigation is most likely to yield the underlying diagnosis?

DAT scan 11%

Anti-GM1b antibodies 9%

Borrelia burgdorferi serology 5%

Urine porphybilinogen	14%
Serum heavy metals	61%

The patient has presented with extrapyramidal signs in the context of a HIV-negative intravenous drug user with alcohol excess. The differential causes for Parkinsonism should be considered: idiopathic, genetic, trauma, vascular etc.

Urinary porphobilinogen is a diagnostic test for acute intermittent porphyria (AIP), typically presenting with significant abdominal pain, urinary symptoms and a peripheral neuropathy. Anti-GM1b are antibodies typically present in multifocal motor neuropathy with conduction block, an immune disorder affecting motor peripheral nerves alone. Heavy metal poisoning must be considered in a young patient with Parkinsonism, in addition to the MPTP toxins found in classic cases of intravenous drug users in 1980s California. This patient is vulnerable to metals such as manganese, which is often found in 'dirty' preparation of intravenous heroin from Eastern Europe and produces extrapyramidal symptoms similar to idiopathic Parkinson's disease. Similar symptoms should be suspected in heavy industry workers.

Discuss (3) | Improve

Next question

Heavy metal poisoning

Heavy metal poisoning or heavy metal toxicity is the accumulation of heavy metals (those with a high density) within the soft tissues of the body. It can be acute or chronic and may be due to ingestion, inhalation or absorption of the metals through the skin or mucous membranes. The metals most commonly linked to poisoning are lead, mercury, arsenic and cadmium, but iron, thallium and bismuth are also metals that may be implicated.

Epidemiology

- Heavy metal poisoning is very rare in the UK
- The incidence of lead poisoning has fallen in affluent countries following the removal of lead paint.

Causes

- Lead is the most common heavy metal to cause poisoning- this is usually due to lead paint or water pipes.

- Ingestion of disc batteries by small children can cause heavy metal poisoning due to mercury, manganese or cadmium content.
- Industrial exposure is another cause of heavy metal poisoning
- Criminal poisoning has been documented using lead and other heavy metals.

The symptoms of heavy metal poisoning depend on the heavy metal involved but almost all metals will cause fatigue, nausea and vomiting. In these notes, we will look at the symptoms and signs of arsenic, lead, mercury and cadmium poisoning as these are the most commonly encountered.

Arsenic Poisoning

Patients with acute poisoning present with:

- garlic-like odour
- hypersalivation
- nausea, vomiting and diarrhoea, abdominal pain
- oral burns, gastrointestinal bleeding
- shortness of breath
- muscle spasms
- renal tubular acidosis
- cardiomyopathy
- jaundice
- intestinal haemorrhage
- seizure
- coma

Patients suffering chronic poisoning may present with:

- hardened patches of skin
- hyperpigmentation of the skin
- anorexia, weight loss
- weakness
- muscle aches
- chills
- polyneuritis, peripheral neuropathy
- ataxia

Signs:

- oedema- subcutaneous and pulmonary
- Mee's lines (transverse white lines across fingernails)
- jaundice
- hypotension

- exfoliative dermatitis
- haemolysis
- ventricular arrhythmia

Lead Poisoning

Patients with acute poisoning present with:

- abdominal pain
- vomiting
- encephalopathy- mania, delirium, seizures
- jaundice
- lethargy

Patients suffering chronic poisoning may present with:

- mild abdominal pain, constipation
- weight loss
- headaches
- aggression and antisocial behaviour
- subfertility- this is due to damage to reproductive organs caused directly by increased lead levels
- motor peripheral neuropathy
- wrist drop, Carpal tunnel syndrome
- autonomic dysfunction

Signs:

- blue discolouration of gums
- anaemia
- behavioural issues- most marked in children with lead over-exposure
- loss of coordination and acquired skills
- loss of reflexes and weakened extensor muscles in adults

Mercury Poisoning

Patients with acute poisoning present with:

- shortness of breath- due to acute pneumonitis with or without adult respiratory distress syndrome (ARDS)
- flu-like illness
- myalgia
- gastrointestinal upset- vomiting, diarrhoea
- peripheral neuropathy

- involuntary movements (chorea), tremor, seizures, cerebellar ataxia

Patients suffering chronic poisoning may present with:

- personality changes and irritability
- headaches
- peripheral neuropathy
- memory problems
- ataxia
- respiratory problems- pneumonitis and ARDS
- Mad Hatter Syndrome (erethism)- a behavioural syndrome characterised by memory loss, excessive shyness, increased excitability and insomnia.

Signs:

- abnormal movements
- dysarthria
- visual field defects- narrowed visual fields
- acrodynia
- erythema
- hyperaesthesia

Cadmium Poisoning

Symptoms:

- metallic taste
- increased salivation
- nausea, vomiting, diarrhoea
- fatigue and headaches
- shortness of breath- secondary to pulmonary oedema and progressive emphysema

Signs:

- hypersalivation
- yellowing of teeth
- cyanosis- may be peripheral or central depending on the severity of lung involvement.
- tachycardia
- anaemia
- proteinuria

Investigations:

- full history: include occupational history and information about the age of the patient's house and where their water is supplied from.

- examination
- FBC: microcytic anaemia in lead and cadmium poisoning
- blood film: basophilic stippling in arsenic and lead poisoning
- blood levels may be taken for mercury and lead levels
- urine levels may be done for mercury and arsenic levels
- Chest X-ray- may show oedema. May also show radiodense pulmonary emboli in mercury injection.
- long-bone X-rays may show 'lead bands' (metaphyseal horizontal lines due to a failure of the bone to remodel) in children with chronic lead over-exposure.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 24-year-old woman presents to the emergency department following a paracetamol overdose. She advises you that she took 20 x 500mg tablets 26 hours ago. Her body weight is 62kg. On examination there is no evidence of decompensated liver failure, she is not confused, and her abdomen is soft and non-tender.

Blood results are as follows:

Hb	122 g/L	Male: (135-180) Female: (115 - 160)
Platelets	164 * 10^9/L	(150 - 400)
WBC	8.2 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	72 µmol/L	(55 - 120)

Bilirubin	14 µmol/L	(3 - 17)
ALP	66 u/L	(30 - 100)
ALT	28 u/L	(3 - 40)
Î³GT	52 u/L	(8 - 60)
Albumin	46 g/L	(35 - 50)

INR	6.2	(0.9-1.1)
Prothrombin time	82 secs	(12-16 secs)
Activated partial thromboplastin time (APTT)	28 secs	(25-35 secs)
Fibrinogen	4.5 g/L	(2 - 4)

Paracetamol level at 26 hours	Awaited

How will you manage this patient?

- Activated charcoal

- [ ] Await paracetamol level results
- [ ] Discuss with the liver transplant unit
- [ ] Gastric lavage
- [ ] Start N-acetylcysteine

Submit answer

Reference ranges

Score: **15.1%**

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Bilirubin	14 µmol/L	(3 - 17)
ALP	66 u/L	(30 - 100)
ALT	28 u/L	(3 - 40)
Î³GT	52 u/L	(8 - 60)
Albumin	46 g/L	(35 - 50)

INR	6.2	(0.9-1.1)
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Activated partial thromboplastin time (APTT)	28 secs	(25-35 secs)
Fibrinogen	4.5 g/L	(2 - 4)

Paracetamol level at 26 hours	Awaited

How will you manage this patient?

Activated charcoal 1%

Await paracetamol level results	9%
Discuss with the liver transplant unit	16%
Gastric lavage	0%
Start N-acetylcysteine	73%

Paracetamol overdose: if presentation > 24 hours after an overdose start acetylcysteine if the patient is jaundiced, has hepatic tenderness or an elevated ALT

Important for me Less important

Start N-acetylcysteine is correct. Acetylcysteine should be started immediately in all patients if it is thought that 150 mg/kg or more paracetamol (or an unknown amount) has been ingested as an acute overdose (i.e. all doses taken within one hour) or if they are symptomatic with jaundice or hepatic tenderness. The patient in this case has ingested 10,000 mg / 62 kg = 161 mg/kg. She should therefore start N-acetylcysteine immediately. If the patient is asymptomatic and has ingested less than 150 mg/kg, then TOXBASE recommends waiting for blood results before considering treatment with N-acetylcysteine.

Start or continue treatment with acetylcysteine if:

- ALT is above the upper limit of normal, or
- INR is greater than 1.3 (in the absence of another cause, e.g. warfarin), or
- Paracetamol concentration is detectable

Activated charcoal is incorrect. Activated charcoal can be considered if the paracetamol has been ingested within one hour of presentation.

Await paracetamol level results is incorrect. Since the patient has ingested > 150 mg/kg of paracetamol she should be started on acetylcysteine immediately. Blood tests including paracetamol level are then monitored serially to determine the ongoing need for N-acetylcysteine treatment.

Discuss with the liver transplant unit is incorrect. There are no indications for liver transplant as per the King's college hospital criteria which includes:

- Arterial pH < 7.3 (24 hours after ingestion)
- All of the following: prothrombin time > 100 seconds, creatinine > 300 µmol/l, and grade III or IV encephalopathy

Gastric lavage is incorrect. Gastric lavage is not recommended due to concerns over perforation.

This was a previously widely favoured method that has now been all but abandoned due to a lack of evidence of efficacy and the risk of complications.

Discuss (4) Improve

Next question

Paracetamol overdose: management

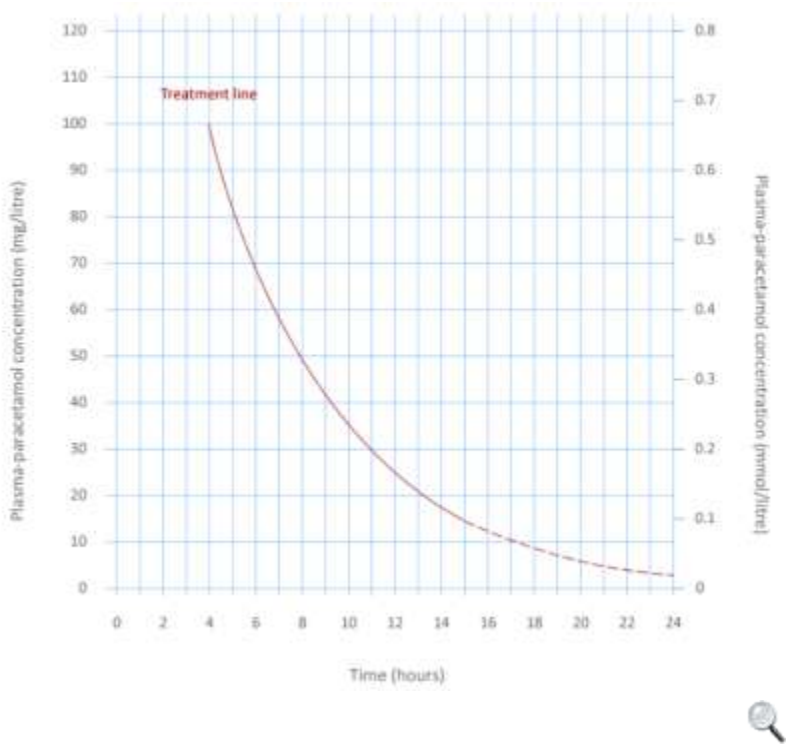
The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

AI + Q 123

Next question

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

Medicines and Healthcare products Regulatory Agency

3 3

2012 Paracetamol overdose update

Suggest link Report broken link

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A 44-year-old man presents to the emergency department complaining of breathlessness. He reports that he was leaving a restaurant because he was starting to feel lightheaded, developed a headache and was breathless.

An ABG done on admission demonstrated carbon monoxide at 26%.

Three hours later he feels tired and weak, but his headache and lightheadedness have improved since admission. He has a past medical history of asthma which he reports to be well controlled, rarely needing his blue inhaler. He smokes 15-ciagarettes per day and has a 23 pack-year history. He works as a teacher in central London. On examination, he is receiving 15L of oxygen via a non-rebreather mask. His chest is clear on auscultation. Neurological examination is normal. An ECG demonstrates no ischaemic changes.

Observations:

Saturations	100%
Respiratory rate	16/min
Blood pressure	126/87mmHg
Heart rate	84/min
Temperature	36.8°C

What is the most appropriate management plan?

- Wean off oxygen guided by standard pulse oximetry saturation levels
- Prescribe IV mannitol
- Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%
- Refer urgently for hyperbaric oxygen treatment
- Arrange urgently for non-invasive ventilation

Submit answer

Reference ranges

Score: **15.1%**

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An ABG done on admission demonstrated carbon monoxide at 26%.

Three hours later he feels tired and weak, but his headache and lightheadedness have improved since admission. He has a past medical history of asthma which he reports to be well controlled, rarely needing his blue inhaler. He smokes 15-ciagarettes per day and has a 23 pack-year history. He works as a teacher in central London. On examination, he is receiving 15L of oxygen via a non-rebreather mask. His chest is clear on auscultation. Neurological examination is normal. An ECG demonstrates no ischaemic changes.

Observations:

Saturations	100%
Respiratory rate	16/min
Blood pressure	126/87mmHg
Heart rate	84/min
Temperature	36.8°C

What is the most appropriate management plan?

Wean off oxygen guided by standard pulse oximetry saturation levels	4%
Prescribe IV mannitol	1%
Maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%	73%
Refer urgently for hyperbaric oxygen treatment	21%
Arrange urgently for non-invasive ventilation	1%

The answer is to maintain high-flow oxygen until asymptomatic or carbon monoxide levels are <10%. Weaning off oxygen is not appropriate to be guided by pulse oximetry as standard pulse oximetry will detect carbon monoxide bound haemoglobin as well and are therefore falsely reassuring. IV mannitol is indicated if cerebral oedema is anticipated, such as in severe cases, or

established. Hyperbaric oxygen again also reserved for severe cases or pregnancy, and non-invasive ventilation has no role in carbon monoxide poisoning.

Source:
'Carbon Monoxide Poisoning.' BMJ Best Practice. N.p., 06 July 2015.

Discuss (7) | Improve

Next question >

Carbon monoxide poisoning

Carbon monoxide has a high affinity for haemoglobin and myoglobin resulting in a left-shift of the oxygen dissociation curve and tissue hypoxia. There are approximately 50 per year deaths from accidental carbon monoxide poisoning in the UK.

Pathophysiology

- carbon monoxide binds readily to haemoglobin, forming carboxyhaemoglobin → reduced oxygen-carrying capacity
- in carbon monoxide poisoning the oxygen saturation of haemoglobin decreases leading to an early plateau in the oxygen dissociation curve

Questions may hint at badly maintained housing e.g. student houses.

Features of carbon monoxide toxicity

- headache: 90% of cases
- nausea and vomiting: 50%
- vertigo: 50%
- confusion: 30%
- subjective weakness: 20%
- severe toxicity: 'pink' skin and mucosae, hyperpyrexia, arrhythmias, extrapyramidal features, coma, death

Investigations

- pulse oximetry may be falsely high due to similarities between oxyhaemoglobin and carboxyhaemoglobin
- therefore a venous or arterial blood gas should be taken
- typical carboxyhaemoglobin levels
  - < 3% non-smokers
  - < 10% smokers

    - 10 - 30% symptomatic: headache, vomiting
    - > 30% severe toxicity
- an ECG is a useful supplementary investgation to look for cardiac ischaemia

Management

- patients with suspected carbon monoxide poisoning should be assessed in the emergency department
- 100% high-flow oxygen via a non-rebreather mask
    - from a physiological perspective, this decreases the half-life of carboxyhemoglobin (COHb)
    - should be administered as soon as possible, with treatment continuing for a minimum of six hours
    - target oxygen saturations are 100%
    - treatment is generally continued until all symptoms have resolved, rather than monitoring CO levels
- hyperbaric oxygen
    - due to the small number of cases the evidence base is limited, but there is some evidence that long-term outcomes may be better than standard oxygen therapy for more severe cases
    - therefore, discussion with a specialist should be considered for more severe cases (e.g. levels > 25%)
    - in 2008, the Department of Health publication 'Recognising Carbon Monoxide Poisoning' also listed loss of consciousness at any point, neurological signs other than headache, myocardial ischaemia or arrhythmia and pregnancy as indications for hyperbaric oxygen

AI + Q 123

Next question >

B I A T T

Textbooks

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Media

Carbon Monoxide Poisoning

Kloss and Bruce - YouTube 3 2

Report broken media

Score: **18.1%**

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A 23-year-old woman is admitted to the emergency department with agitation and she refuses to answer any questions. Her partner is with her and reports she has become increasingly agitated throughout the day. He states she has no past medical history but is on some tablets for her mood which her GP has recently changed. You find that she is taking sertraline and mirtazapine, but the patient and her partner are unable to tell you dosages.

The patient allows you to examine her briefly. No abnormality is found on cardio-respiratory and abdominal examination. You note her pupils are dilated, her reflexes are brisk and myoclonus is present. Her observations are shown below:

Temperature	38.8ºC
Heart rate	110/min
Blood pressure	178/102mmHg
Respiratory rate	25/min
Oxygen saturation	98% on air

Blood tests are in progress. You prescribe some intravenous fluids and lorazepam to calm the patient.

Given the diagnosis, what other treatment should the patient receive?

- Dantrolene
- Cyproheptadine
- Bromocriptine
- Co-amoxiclav
- Procyclidine

Submit answer

Reference ranges

Score: **15.1%**

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Heart rate	110/min
Blood pressure	178/102mmHg
Respiratory rate	25/min
Oxygen saturation	98% on air

Blood tests are in progress. You prescribe some intravenous fluids and lorazepam to calm the patient.

Given the diagnosis, what other treatment should the patient receive?

Dantrolene	17%
Cyproheptadine	75%
Bromocriptine	2%
Co-amoxiclav	0%
Procyclidine	6%

Treatment of severe serotonin syndrome- cyproheptadine (5-HT2 receptor antagonist)

Important for me Less important

The patient is presenting with serotonin syndrome. The clues in the history that lead to the diagnosis are the fact that she is on 2 serotonergic agents, she has autonomic excitability (tachycardia, hypertension etc), and neurological features such as hyperreflexia. The differential diagnosis could be neuroleptic malignant syndrome which also presents with autonomic excitability, however, the patient is not taking a neuroleptic and the neurological features would be different (hyporeflexia is expected).

Treatment of serotonin syndrome is usually supportive but in severe cases, cyproheptadine can be used and so option 2 is correct.

Dantrolene is a muscle relaxant that acts via inhibiting calcium release in muscle tissue and is used for the treatment of neuroleptic malignant syndrome.

Bromocriptine is a dopamine agonist, also used in the treatment of neuroleptic malignant syndrome.

Co-amoxiclav is an antibiotic that can be used for numerous infections. Although the patient is febrile, there is no other information from the history or examination to suggest an infection, therefore is not the correct answer.

Procyclidine is an anticholinergic used for acute dystonia.

Discuss (1) | Improve

Next question >

Serotonin syndrome ★

Causes

- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

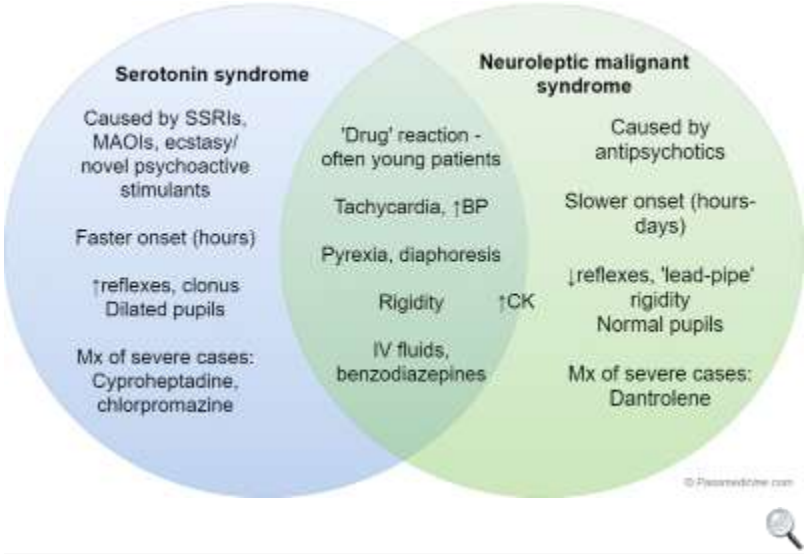
Features

- neuromuscular excitation
  - hyperreflexia

    - myoclonus
    - rigidity
- autonomic nervous system excitation
    - hyperthermia
    - sweating
- altered mental state
    - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe

1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link

Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

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Score: **18.1%**

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A 66-year-old gentleman was admitted for an elective aortic valve replacement. His past medical history included a non-ST elevation myocardial infarction three years prior, as well as hypertension and hypercholesterolaemia. He also suffered a deep vein thrombosis 16 years ago after open reduction and internal fixation of a tibial fracture requiring treatment with warfarin postoperatively. There was no family history of note. He was currently taking aspirin 75mg OD, clopidogrel 75mg OD, ramipril 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg ON and lansoprazole 15mg OD.

Two days prior to the operation the patient was commenced on an intravenous heparin infusion which was continued perioperatively. The operation was deemed successful and the infusion was discontinued. He was commenced on enoxaparin 40mg OD sc as prophylaxis for venous thromboembolism postoperatively. He was otherwise making a good post operative recovery and was already mobilising readily. He complained of no chest pain or shortness of breath with no syncopal episodes. He resumed a full and normal diet and his bowel habits were normal. There was no evidence of bleed or any other adverse symptoms.

Routine post operative bloods at day 5 were as follows:

Hb	110 g/l
MCV	81 fl
Haematocrit	0.36
Platelets	28* 10^9/l
WBC	9.0 * 10^9/l
PT	36 seconds
APTT	86 seconds
D-dimer	136 ng/mL
Fibrinogen	236 g/L

Na^+	142 mmol/l
K^+	4.5 mmol/l
Urea	6.3mmol/l
Creatinine	77 µmol/l
Bilirubin	17 µmol/l
ALP	101 u/l

ALT	16 u/l
Albumin	39 g/l

Blood tests at day 2 were as follows:

Hb	116 g/l
Platelets	161 * 10^9/l
WBC	10.0 * 10^9/l

What is the single most appropriate management step?

- ○ Stop heparin and commence treatment with vitamin K ×
- ○ Continue with enoxaparin ×
- ○ Stop all anticoagulation until platelet count is >100 x 10^9/l ×
- ○ Stop enoxaparin and commence danaparoid ×
- ○ Stop enoxaparin and commence warfarin ×

Submit answer

Reference ranges

Score: **15.1%**

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A 66-year-old gentleman was admitted for an elective aortic valve replacement. His past medical history included a non-ST elevation myocardial infarction three years prior, as well as hypertension and hypercholesterolaemia. He also suffered a deep vein thrombosis 16 years ago after open reduction and internal fixation of a tibial fracture requiring treatment with warfarin postoperatively. There was no family history of note. He was currently taking aspirin 75mg OD, clopidogrel 75mg OD, ramipril 5mg OD, bisoprolol 2.5mg OD, atorvastatin 20mg ON and lansoprazole 15mg OD.

Two days prior to the operation the patient was commenced on an intravenous heparin infusion which was continued perioperatively. The operation was deemed successful and the infusion was discontinued. He was commenced on enoxaparin 40mg OD sc as prophylaxis for venous thromboembolism postoperatively. He was otherwise making a good post operative recovery and was already mobilising readily. He complained of no chest pain or shortness of breath with no syncopal episodes. He resumed a full and normal diet and his bowel habits were normal. There was no evidence of bleed or any other adverse symptoms.

Routine post operative bloods at day 5 were as follows:

Hb	110 g/l
MCV	81 fl
Haematocrit	0.36
Platelets	28* 10^9/l
WBC	9.0 * 10^9/l
PT	36 seconds
APTT	86 seconds
D-dimer	136 ng/mL
Fibrinogen	236 g/L

Na^+	142 mmol/l
K^+	4.5 mmol/l
Urea	6.3mmol/l
Creatinine	77 µmol/l
Bilirubin	17 µmol/l
ALP	101 u/l

ALT	16 u/l
Albumin	39 g/l

Blood tests at day 2 were as follows:

Hb	116 g/l
Platelets	161 * 10^9/l
WBC	10.0 * 10^9/l

What is the single most appropriate management step?

- Stop heparin and commence treatment with vitamin K — 2%
- Continue with enoxaparin — 9%
- Stop all anticoagulation until platelet count is >100 x 10^9/l — 7%
- **Stop enoxaparin and commence danaparoid — 76%**
- Stop enoxaparin and commence warfarin — 5%

The diagnosis is strongly suggestive of heparin induced thrombocytopaenia type 2, given the treatment with intravenous heparin, the platelet count in the absence of other haematological abnormalities, the absence of clinical signs of haemorrhage and the onset of low platelet count at day 5 post operatively. This patient is at very high risk of an arterial or venous thrombotic event and needs urgent anticoagulation. Warfarin should not be used alone and there is a 10% cross-reactivity with LMWH, and so of the options available anticoagulation with danaparoid is the most suitable option.

Discuss (8) | Improve

Next question >

Heparin ★

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin

forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy

- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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A 45-year-old man presents to the emergency department complaining of palpitations. He states that these started suddenly 2 hours ago. He has no past medical history and takes no regular medication. An ECG (electrocardiogram) is done and shows atrial flutter with 2:1 block

His observations are the following

- Sats 97% on air
- Heart rate 150 bpm
- Respiratory rate 22/min
- Blood pressure 105/78 mmHg
- Temperature 36.8ºC

Which of the following treatments should be avoided?

- Electrical cardioversion
- Atenolol
- Diltiazem
- Digoxin
- Flecainide

Submit answer

Reference ranges

Score: **15.1%**

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His observations are the following

- Sats 97% on air
- Heart rate 150 bpm
- Respiratory rate 22/min
- Blood pressure 105/78 mmHg
- Temperature 36.8ºC

Which of the following treatments should be avoided?

Electrical cardioversion	10%
Atenolol	13%
Diltiazem	17%
Digoxin	12%
Flecainide	47%

You risk causing 1:1 conduction if you give flecainide in atrial flutter

Important for me Less important

Flecainide slows the rate of atrial flutter which can 1:1 conduction and cardiovascular compromise. It can also cause 'organisation' of atrial fibrillation turning it into atrial flutter with 1:1 conduction.

Class IA and class IC antiarrhythmic drugs should not be given to treat atrial flutter unless patients are first pre-treated with a drug that blocks AV (atrioventricular) node conduction such as a beta blocker or calcium channel blocker.

All of the other options can be used to treat atrial flutter

- Cardioversion should be used first line

- In patients who are not suitable for cardioversion beta blockers and calcium channel blockers can be used to slow AV node conduction
- Digoxin used to be first line treatment for ventricular rate control in flutter. Now it is used mostly in patients with heart failure alongside a beta blocker. It is also used in patients whose rate has not been adequately controlled by a calcium channel blocker, a beta blocker or both.

Management of Atrial flutter

- Is similar to that of atrial fibrillation although medication may be less effective
- Atrial flutter is more sensitive to cardioversion however so lower energy levels may be used
- Radiofrequency ablation of the tricuspid valve isthmus is curative for most patients

Discuss (1) | Improve

Next question >

Flecainide ★

Flecainide is a Vaughan Williams class 1c antiarrhythmic. It slows conduction of the action potential by acting as a potent sodium channel blocker (specifically the Nav1.5 sodium channels). This may be reflected by widening of the QRS complex and prolongation of the PR interval.

The Cardiac Arrhythmia Suppression Trial (CAST, 1989) investigated the use of agents to treat asymptomatic or mildly symptomatic premature ventricular complexes (PVCs) post myocardial infarction. The hypothesis was that this would reduce deaths from ventricular arrhythmias. Flecainide was actually shown to increase mortality post-myocardial infarction and is, therefore, contraindicated in this situation.

Indications

- atrial fibrillation
- SVT associated with accessory pathway e.g. Wolf-Parkinson-White syndrome

Contraindications

- post myocardial infarction
- structural heart disease: e.g. heart failure
- sinus node dysfunction; second-degree or greater AV block
- atrial flutter

Adverse effects

- negatively inotropic
- bradycardia
- proarrhythmic
- oral paraesthesia
- visual disturbances

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF

1 2

Flecainide

Suggest link

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Score: **18.1%**

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A 31-year-old man is brought to the Emergency Department from a nightclub. He is accompanied by a friend who tells you he has taken the street drug NRG-1. The patient is agitated and difficult to assess as he will not stay on the trolley. He is sweating profusely. Formal assessment is impossible but he is obviously confused and appears to be hallucinating. Examination shows a heart rate of 139bpm with a blood pressure of 158/105mmHg. Heart sounds are rapid but with no murmurs. The chest is clear to auscultation and oxygen saturations are 99% on air but his respiratory rate is 36/min. He is globally hypertonic with marked clonus in the lower limbs. Tympanic temperature is 40.6°C.

Blood results show:

Haemoglobin	129g/L	Sodium	119mmol/L	Creatine Kinase	1895ng/mL
Haematocrit	0.29	Potassium	5.5mmol/L	CRP	8.9mg/L
White cells	15.6x10^9/L	Chloride	90mmol/L		
Neutrophils	11.7x10^9/L	Urea	7.2mmol/L		
Platelets	303x10^9/L	Creatinine	100µmol/L		

Urine is dilute but contains blood, protein and ketones on dipstick analysis

Which one of the following interventions is the most appropriate in the first instance?

- Rapid intravenous infusion of 0.9% sodium chloride with 1g IV paracetamol
- Rapid intravenous infusion of 3% sodium chloride with 1g IV paracetamol
- Slow intravenous infusion of 3% sodium chloride with 10mg IV midazolam
- Slow intravenous infusion of 3% sodium chloride with 1g IV paracetamol and oral cyproheptadine 12mg
- Intravenous methylene blue (methylthioninium chloride) 1mg/kg over 5 minutes

Submit answer

Reference ranges

Score: **15.1%**

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Platelets	303x10^9/L	Creatinine	100µmol/L		

Urine is dilute but contains blood, protein and ketones on dipstick analysis

Which one of the following interventions is the most appropriate in the first instance?

Rapid intravenous infusion of 0.9% sodium chloride with 1g IV paracetamol	18%
Rapid intravenous infusion of 3% sodium chloride with 1g IV paracetamol	8%
Slow intravenous infusion of 3% sodium chloride with 10mg IV midazolam	22%
Slow intravenous infusion of 3% sodium chloride with 1g IV paracetamol and oral cyproheptadine 12mg	46%
Intravenous methylene blue (methylthioninium chloride) 1mg/kg over 5 minutes	6%

NRG-1, a synthetic cathinone, can cause agitation, hyponatraemia and serotonin syndrome. Treatment is with benzodiazepines, cooling and hypertonic saline if hyponatraemic. Patients may require intubation and paralysis to control hyperpyrexia

Important for me Less important

Hyperpyrexia is a consequence of muscle activity, not hypothalamic dysregulation so paracetamol is not useful. Indeed, if multiorgan failure develops, paracetamol is not conjugated and excreted safely, thus paracetamol toxicity may worsen the picture. Intravenous benzodiazepines are the mainstay in reducing agitation and helping to control muscle activity and this should be commenced in the short term. Given the concurrent hyponatraemia and elevated CK slow fluids with hypertonic saline is advised.

External cooling manoeuvres such as ice baths may be tried but when hyperpyrexia rises above 40°C it is safer to anaesthetise and paralyse patients and this patient is likely to warrant this.

Cyproheptadine is a specific 5HT-2A receptor antagonist and may be used in moderate serotonin syndrome. In this case, it is unlikely the patient will be cooperative enough to take it and its effects will be too slow. This option also includes paracetamol making this an incorrect answer.

Methylene blue is an antidote for methaemoglobinaemia; it would be unusual for NRG-1 to cause this although users of the drug often display a blue tinge to their skin for unknown reasons. The methaemoglobin level is not given here so use of methylene blue is not advisable.

Discuss (5) Improve

Next question ›

Cathinone toxicity ★

NRG-1 is a synthetic cathinone drug which is increasingly used recreationally. Pharmacologically it is a derivative of phenylpropanone which is a naturally occurring psychotrope in khat (*Catha edulis*).

Synthetic cathinones became increasingly popular in the last ten years as an alternative to ecstasy since they were cheaper, easier to produce and initially were unrestricted. As legislation changes, chemical substitutions are made to molecular moieties to create similar drugs to avoid restrictions. All exert their effect by increasing synaptic concentrations of noradrenaline, dopamine and serotonin, giving users the sensation of euphoria, detachment and wellbeing as well as upregulation of the sympathetic system.

Toxicity is often seen due to lack of regulation of constituents and active ingredients. Tachycardia and hypertension may be seen due to the sympathomimetic effects of the drug and in some cases myocardial ischaemia can be seen. In the majority of cases of toxicity, however, similar to ecstasy toxicity, hyponatraemia and serotonin syndrome are seen. Hyponatraemia occurs as a

consequence of significant water intake to reduce body temperature. Serum sodium levels may be markedly low and patients may present seizing.

If there is evidence of neurological compromise with an accompanying hyponatraemia, rapid correction of sodium is recommended with infusion of 3% saline solution at a maximum rate of 1ml/kg/hour. 0.9% saline solution is not recommended in patients with hyponatraemia and agitation due to the risk of worsening the hyponatraemia. Serotonin syndrome is due to massive flooding of synapses with liberated serotonin and causes agitation, confusion, muscle hyperactivity with fasciculations, hypertonia and clonus. Creatine kinase and white cell counts are often raised and body temperature may be extremely high.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 26 year old male is admitted to hospital with difficulty in breathing, wheeze and a cough productive of green sputum. He is assessed and determined to have a community acquired lower respiratory tract infection with biochemical and radiological evidence lending weight to this diagnosis. His medical history is significant for capricious asthma and he has been admitted to intensive care previously and ventilated. Currently he is taking salbutamol and formoterol/budesonide inhalers at their maximum doses, montelukast, aminophylline and omalizumab (Xolair) monthly subcutaneous injections.

Which of the following antibiotics is best avoided in this patients treatment?

- Amoxicillin
- Azithromycin
- Ciprofloxacin
- Doxycycline
- Gentamicin

Submit answer

Reference ranges

Score: **15.1%**

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Which of the following antibiotics is best avoided in this patients treatment?

Co-administration of aminophylline and ciprofloxacin can cause significant toxicity and should be avoided

Important for me Less important

This question is about drugs which interact via their effect on hepatic cytochrome metabolising enzymes. Pharmacokinetically speaking, any drug has the potential to affect how another is processed, metabolised or excreted but only some have a relevant clinical effect.

In the above vignette we are concerned about a newly commenced antibiotic affecting one of the patients regular medications and causing a deleterious effect. Salbutamol and formoterol are beta agonist drugs which have their own side effects of adrenergic updrive but they rarely have any clinically relevant interactions. Similarly, the inhaled steroid budesonide is seldom affected by other co-administered drugs to a vast degree. Omalizumab is a monoclonal antibody to unbound human IgE and is used as an adjunct treatment in atopic asthma in steroid resistant patients who display extreme histamine sensitivity and elevated IgE levels. It has few clinical interactions. Aminophylline derivatives are moderately active bronchodilators which work two-fold; by inhibiting phosphodiesterases and hence ultimately dampening inflammatory cytokines, and also

by acting as an antagonist at adenosine receptors in the heart, brain and lung. It is metabolised extensively by the liver, predominantly by the enzyme 1A2 and has a narrow therapeutic window with elimination pathways becoming saturated even at therapeutic doses. Any drug which inhibits cytochrome enzyme 1A2 can rapidly lead to aminophylline toxicity.

Ciprofloxacin in particular, is a strong inhibitor of enzyme 1A2 causing a decrease in function of at least 80% and an average rise in serum concentrations of metabolising substrate of five-fold. Co-administration of ciprofloxacin with aminophylline is extremely likely to cause toxicity.

The use of macrolide antibiotics with aminophylline is contentious. Most macrolides actually induce activity of 1A2 leading to a reduction in aminophylline levels (similar to the effect of long term smoking). Both aminophylline and macrolide antibiotics however tend to predispose to prolongation of myocardial repolarisation time and there is a risk of ventricular arrhythmia. Azithromycin only poses a theoretical risk rather than an observed one and dose reduction and drug monitoring of aminophylline levels is appropriate if the two drugs must be used together. Erythromycin is best avoided.

Doxycycline use with aminophylline can cause theoretical fluctuations in serum aminophylline levels but regular monitoring of drug levels is sufficient to avoid toxicity.
Neither amoxicillin nor gentamicin have clinically relevant drug interactions with aminophylline.

Discuss (8) Improve

Next question

P450 drug interactions: more detail

Whilst you are expected to know in broad terms what are the main inhibitors and inducers of the P450 system it is unlikely that you will be asked detailed questions about the individual enzyme systems.

It is worthwhile noting that the most important and common reason for drug interactions is the P450 **CYP3A4 system**.

The table below shows the main enzyme systems that are affected by common drugs. There is clearly a lot of overlap within the various P450 enzymes.

P450 system	Substrates	Inhibitors	Inducers
CYP3A4	Macrolides Antiretrovirals Calcium channel blockers	Macrolides Protease inhibitors (including ritonavir) Imidazoles	Carbamazepine Phenytoin Phenobarbitone Rifampicin St John's Wort
CYP2D6	Tricyclic antidepressants Antipsychotics	SSRIs Ritonavir	
CYP2C9	Warfarin Sulfonylureas	Imidazoles Amiodarone Sodium valproate	Rifampicin
CYP1A2	Theophylline	Ciprofloxacin	Smoking Omeprazole
CYP2E1	Alcohol		Chronic alcohol Isoniazid

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 27-year-old patient has been asked to attend the emergency department 2 weeks post colectomy and stoma formation due to the following blood results:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	412 * 10^9/L	(150 - 400)
WBC	10 * 10^9/L	(4.0 - 11.0)
Na^+	147 mmol/L	(135 - 145)
K^+	4.9 mmol/L	(3.5 - 5.0)
Urea	16.9 mmol/L	(2.0 - 7.0)
Creatinine	244 µmol/L	(55 - 120)
CRP	65 mg/L	(< 5)

She has been struggling to manage her stoma due to high output of between 1-2litres of liquid stool per day. She has been using rehydration salts and loperamide at home.

Abdominal x-ray: unremarkable appearances
Stool culture: negative

What further treatment would be most appropriate to reduce her high-output diarrhoea?

- Bismuth subsalicylate
- IV fluids
- Morphine sulphate
- Octreotide
- Vancomycin

Submit answer

Reference ranges

Score: **15.1%**

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She has been struggling to manage her stoma due to high output of between 1-2litres of liquid stool per day. She has been using rehydration salts and loperamide at home.

Abdominal x-ray: unremarkable appearances
Stool culture: negative

What further treatment would be most appropriate to reduce her high-output diarrhoea?

- Bismuth subsalicylate — 19%
- IV fluids — 2%
- Morphine sulphate — 13%
- **Octreotide** — 63%
- Vancomycin — 2%

Octreotide may reduce high output diarrhoea following ileostomy or colectomy. It can be given by continuous subcutaneous infusion

Important for me Less important

Octreotide is correct. This can be given via a continuous subcutaneous infusion in the management of high-output stoma diarrhoea.

Bismuth subsalicylate is incorrect. It is usually used for symptoms of dyspepsia and is occasionally used in the management of travellers' diarrhoea. There is no evidence to support its use in the reduction of diarrhoea as a result of a high-output stoma.

Morphine sulphate is incorrect. Although morphine and other opiate-class medications can cause constipation as a side effect, they are not used routinely in the management of high-output stomas.

IV fluids is incorrect. Using IV fluids to manage acute kidney injury would be entirely appropriate, however, it would not have any effect on the ongoing diarrhoea itself.

Vancomycin is incorrect, this would be the treatment for *C. difficile* diarrhoea, however, in this case, stool samples were unremarkable.

Discuss (5) | Improve

Next question >

Octreotide ★

Overview

- long-acting analogue of somatostatin
- somatostatin is released from D cells of pancreas and inhibits the release of growth hormone, glucagon and insulin

Uses

- acute treatment of variceal haemorrhage
- acromegaly
- carcinoid syndrome
- prevent complications following pancreatic surgery
- VIPomas
- refractory diarrhoea

Adverse effects

- gallstones (secondary to biliary stasis)

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 28-year-old woman attends the emergency department with reduced consciousness. She has a past medical history of depression which is treated with sertraline. She also has chronic pain (for which she takes tramadol, gabapentin, ibuprofen, and a lidocaine patch) and epilepsy (which is managed with phenytoin).

On examination, her Glasgow Coma Score is 11 (M4, V4, E3). You also note mydriasis, hyperreflexia, and clonus.

What drug is most likely implicated?

- Gabapentin
- Ibuprofen
- Lidocaine
- Phenytoin
- Tramadol

Submit answer

Reference ranges

Score: **15.1%**

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On examination, her Glasgow Coma Score is 11 (M4, V4, E3). You also note mydriasis, hyperreflexia, and clonus.

What drug is most likely implicated?

Gabapentin	10%
Ibuprofen	3%
Lidocaine	7%
Phenytoin	17%
Tramadol	63%

Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome

Important for me Less important

The patient has clinical features of serotonin syndrome.

Tramadol is correct. Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome.

Gabapentin is incorrect. The use of SSRIs with gabapentin can increase the risk of hyponatraemia.

Lidocaine is incorrect. There are no interactions with SSRIs and lidocaine.

Phenytoin is incorrect. The use of sertraline with phenytoin increases the risk of phenytoin toxicity. Phenytoin toxicity manifests predominantly as nausea, and central nervous system dysfunction (particularly confusion, nystagmus, and ataxia), with depressed conscious state, coma, and seizures occurring in more severe cases. Cardiac complications such as arrhythmias and hypotension can also occur. The clinical features, in this case, are more in keeping with serotonin syndrome.

Ibuprofen is incorrect. The use of these two agents increases bleeding risk. It is important to remember that SSRIs have an anti-platelet effect since serotonin is an important transmitter involved in platelet function.

Discuss | Improve

Next question >

Serotonin syndrome ★

Causes

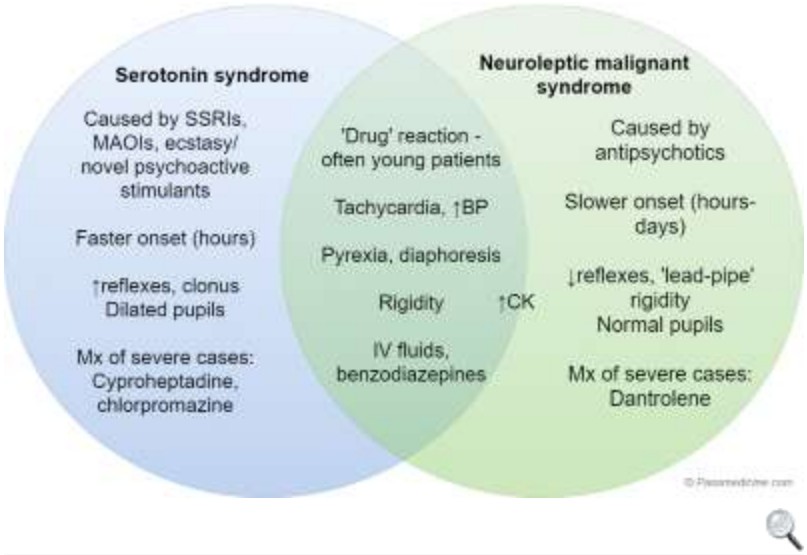
- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

Features

- neuromuscular excitation
  - hyperreflexia
  - myoclonus
  - rigidity
- autonomic nervous system excitation
  - hyperthermia
  - sweating
- altered mental state
  - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

AI + Q 123

Next question

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe 1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

Report broken media

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Back to top

A 27-year-old man is brought to the Emergency Department following a suspected deliberate overdose. On arrival, he is drowsy with a GCS of 13/15 (E3V4M6) and confused. His airway is currently patent. The respiratory rate is 30 breaths per minute although oxygen saturations are 98% on 2L/min oxygen. The chest is clear to auscultation. Heart rate is 108bpm and blood pressure is 96/48mmHg. The ECG shows sinus tachycardia only. He complains of severe abdominal pain and is groaning on examination. There is significant tenderness in the right upper quadrant. Bowel sounds are hyperactive. Neurological examination is difficult but discloses downgoing plantars and reactive, mid-size pupils.

Blood tests show:

Haemoglobin	112g/L	Sodium	131mmol/L	Glucose	17.8mmol
MCV	96fL	Potassium	Haemolysed	Lactate	5.3mmol/L
White cells	15.7x109/L	Urea	15.6mmol/L	pH	7.14
Neutrophils	13.7x109/L	Creatinine	207mol/L	pCO2	2.9kPa
Platelets	278x109/L	ALP	234U/L	pO2	19.2kPa
INR	1.2	ALT	2453U/L	Bicarbonate	9.4mmol/L
PT	14secs	AST	3109U/L	Base excess	-15.4mEq/L
APTT	49.2secs	Bilirubin	36mol/L	Anion gap	30.8

The laboratory comments there is significant haemolysis of the samples.

He has a massive vomit whilst being assessed, of black liquid and fresh blood.

Which of the following drugs is this patient most likely to have overdosed upon?

- Aspirin
- Ferrous sulphate
- Ibuprofen
- Paracetamol
- Venlafaxine

Submit answer

Reference ranges

Score: **15.1%**

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A 27-year-old man is brought to the Emergency Department following a suspected deliberate overdose. On arrival, he is drowsy with a GCS of 13/15 (E3V4M6) and confused. His airway is currently patent. The respiratory rate is 30 breaths per minute although oxygen saturations are 98% on 2L/min oxygen. The chest is clear to auscultation. Heart rate is 108bpm and blood pressure is 96/48mmHg. The ECG shows sinus tachycardia only. He complains of severe abdominal pain and is groaning on examination. There is significant tenderness in the right upper quadrant. Bowel sounds are hyperactive. Neurological examination is difficult but discloses downgoing plantars and reactive, mid-size pupils.

Blood tests show:

Haemoglobin	112g/L	Sodium	131mmol/L	Glucose	17.8mmol
MCV	96fL	Potassium	Haemolysed	Lactate	5.3mmol/L
White cells	15.7x109/L	Urea	15.6mmol/L	pH	7.14
Neutrophils	13.7x109/L	Creatinine	207mol/L	pCO2	2.9kPa
Platelets	278x109/L	ALP	234U/L	pO2	19.2kPa
INR	1.2	ALT	2453U/L	Bicarbonate	9.4mmol/L
PT	14secs	AST	3109U/L	Base excess	-15.4mEq/L
APTT	49.2secs	Bilirubin	36mol/L	Anion gap	30.8

The laboratory comments there is significant haemolysis of the samples.

He has a massive vomit whilst being assessed, of black liquid and fresh blood.

Which of the following drugs is this patient most likely to have overdosed upon?

Aspirin	28%
Ferrous sulphate	31%
Ibuprofen	7%
Paracetamol	30%
Venlafaxine	4%

Severe iron toxicity presents with liver failure, gastrointestinal caustic damage and coagulopathy with raised APTT. Early hyperglycaemia and extensively haemolysed samples may also indicate significant iron burden

Important for me Less important

Iron is a surprisingly toxic drug. When taken in overdose symptoms may be expected to be seen at ingestions of 20mg/kg. 200mg/kg can be expected to be fatal. Symptoms of direct corrosion on the gastrointestinal tract may be seen within six hours of ingestion with abdominal pain, vomiting, diarrhoea and GI bleeding. Stool and vomitus may be black or grey unless frank bleeding predominates. During this interval, a hallmark of iron toxicity is a raised blood glucose; only a few drugs may cause this in overdose. Hypoglycaemia occurs later in presentation due to liver failure. Other early features may include a neutrophil leucocytosis and significant haemolysis of samples due to the high plasma iron burden. Arterial blood gases will show a significant metabolic acidosis with a high anion gap. Lactate may be elevated but not sufficiently to explain the anion gap. Coagulopathy develops rapidly but the APTT tends to be prolonged in iron toxicity compared to the prolongation of prothrombin time in a paracetamol poisoning. In the ensuing hours, iron deposition within the myocardium, brain and liver can be expected to cause rapid cardiac failure, encephalopathy and hepatocellular necrosis with liver failure respectively. Renal failure develops due to hypovolaemia and acute tubular necrosis. Unless treated, death may result from haemorrhagic volume loss or from hepatic failure, although in significant overdose, early death results from respiratory failure due to coma. Treatment for iron overdose is with decontamination of the bowel, including whole bowel irrigation if needed and chelation therapy with desferrioxamine.

Although paracetamol toxicity can be expected to cause fulminant hepatic failure with renal failure and coagulopathy, the INR would be expected to be significantly deranged if this was the culpable drug given the extent of other organ damage. Also hypoglycaemia would be expected due to liver failure.

Aspirin, when taken in significant overdose, causes a characteristic constellation of symptoms including metabolic acidosis (although alkalosis may be seen at lower dose intoxications), convulsions, tinnitus, hyperpyrexia and pulmonary oedema. Often ventricular arrhythmia occurs due to metabolic acidosis and intracellular anaerobic respiration. Rarely coagulopathy develops but usually in association with disseminated intravascular coagulation which is not the case here. Aspirin overdoses may present with hyperglycaemia however and along with iron and aminophylline, these agents should be considered when unexplained hyperglycaemia is seen.

Ibuprofen and other non-steroidals are of low toxicity unless taken in massive overdose. Gastrointestinal upset and occasional GI haemorrhaging may be seen but liver failure and coagulopathy is rare. In massive overdose, central nervous depression with cerebellar signs and global electrolyte imbalances may be seen with blood dyscrasias. Multi-organ failure may develop but individual organ dysfunction is less likely.

Venlafaxine, a noradrenaline and serotonin modulator, may cause serotonin syndrome and sympathomimetic symptoms, including arrhythmia, hyperpyrexia and respiratory failure. Hepatitis is rare.

Discuss (3) | Improve

Next question >

Overdose and poisoning: management

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization with IV bicarbonate • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil The majority of overdoses are managed with supportive care only due to the risk of seizures with flumazenil. It is generally only used with severe or iatrogenic overdoses.

Toxin	Treatment
Tricyclic antidepressants	Management • IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity • arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics
Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine • in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases

Toxin	Treatment
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit
Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

AI + Q 123

Next question ›

B I A T

Textbooks

High-yield textbook

Extended textbook

Links

BNF — 2 1

Emergency treatment of poisoning

Royal College of Physicians — 1 1

2012 Poisoning and self-harm update

Royal College of Physicians — 1 2

2007 Acute poisoning on the medical admissions unit

Suggest link | Report broken link

Score: **18.1%**

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A 21-year-old woman presents to the emergency department after her flatmate found her drowsy in her bedroom. She had been incontinent of urine.

On examination the patient is obtunded with a Glasgow Coma Score of 7 (E1V2M4). The pupils are 7mm bilaterally and sluggishly reactive to light. The heart rate is 133bpm and the blood pressure is 99/65mmHg.

A 12-lead ECG reveals sinus tachycardia with a QT interval of 510ms. The QRS duration is 115ms.

What is the most likely cause for this patient's presentation?

- Amyl nitrate toxicity
- Gamma hydroxybutyric acid toxicity
- Toluene solvent toxicity
- Tricyclic antidepressant toxicity
- Heroin toxicity

Submit answer

Reference ranges

Score: **15.1%**

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A 12-lead ECG reveals sinus tachycardia with a QT interval of 510ms. The QRS duration is 115ms.

What is the most likely cause for this patient's presentation?

Amyl nitrate toxicity	4%
Gamma hydroxybutyric acid toxicity	7%
Toluene solvent toxicity	2%
Tricyclic antidepressant toxicity	83%
Heroin toxicity	3%

Dilated pupils in combination with ECG changes including prolonged QT interval and QRS duration (borderline prolonged in this case) should suggest tricyclic antidepressant toxicity.

Features of toluene toxicity include irritation to the eyes, nose and respiratory tract from inhalation, along with confusion, ataxia, headache, slurred speech and euphoria. Prolongation of the QT interval is not usually associated.

Features of amyl nitrate toxicity include blurred vision, xanthopsia and haemoptysis. It does not typically affect the corrected QT interval.

Features of gamma hydroxybutyric acid (GHB) toxicity include CNS and respiratory depression, hypersalivation, bradycardia and hypotension. It does not typically affect the corrected QT interval.

Heroin toxicity typically results in depression of the respiratory and central nervous systems and pin-point pupils (rather than mydriasis in this case). It does not typically affect the corrected QT interval.

(www.toxbase,org)

Discuss (2) | Improve

Next question >

Tricyclic overdose

Overdose of tricyclic antidepressants is a common presentation to emergency departments. Amitriptyline and dosulepin (dothiepin) are particularly dangerous in overdose.

Early features relate to anticholinergic properties: dry mouth, dilated pupils, agitation, sinus tachycardia, blurred vision.

Features of severe poisoning include:

- arrhythmias
- seizures
- metabolic acidosis
- coma

ECG changes include:

- sinus tachycardia
- widening of QRS
- prolongation of QT interval

Widening of QRS > 100ms is associated with an increased risk of seizures whilst QRS > 160ms is associated with ventricular arrhythmias

Management

- IV bicarbonate
  - first-line therapy for hypotension or arrhythmias
  - indications include widening of the QRS interval >100 msec or a ventricular arrhythmia
- other drugs for arrhythmias

+ class 1a (e.g. quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation
    - class III drugs such as amiodarone should also be avoided as they prolong the QT interval
    - response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in the management of tricyclic-induced arrhythmias

- intravenous lipid emulsion is increasingly used to bind free drug and reduce toxicity
- dialysis is ineffective in removing tricyclics

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 48-year-old farmer is found in a state of distress having collapsed whilst mixing insecticides in his barn. On arrival in the Emergency department, he is confused, salivating excessively, and has been incontinent of urine and faeces. You note noisy breathing, his blood pressure is 90/60 mmHg, with a pulse of 48 beats per minute.

Which of the following is the most appropriate intervention?

- Atropine
- Hyoscine
- Glucagon
- NG activated charcoal
- Noradrenaline

Submit answer

Reference ranges

Score: **15.1%**

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A 48-year-old farmer is found in a state of distress having collapsed whilst mixing insecticides in his barn. On arrival in the Emergency department, he is confused, salivating excessively, and has been incontinent of urine and faeces. You note noisy breathing, his blood pressure is 90/60 mmHg, with a pulse of 48 beats per minute.

Which of the following is the most appropriate intervention?

Atropine	95%
Hyoscine	1%
Glucagon	2%
NG activated charcoal	1%
Noradrenaline	1%

Organophosphate insecticide - atropine

Important for me Less important

The clue here is insecticide exposure, coupled with evidence of muscarinic effects of organophosphate poisoning including excessive secretions, incontinence, hypotension and bradycardia. Time is of the essence, and administration of atropine in this situation may be life-saving.
Pralidoxime is also used once atropine has been administered, although it's additive benefit is debated.

Hyoscine is less potent versus atropine and is mainly used in palliative care to manage pain from the gastrointestinal tract and excessive respiratory secretions. Glucagon is used as a treatment for beta-blocker overdose. Activated charcoal may be of value but is subsidiary to use of anticholinergics. Anticholinergics are also preferred to sympathetic agonists such as noradrenaline.

Organophosphate insecticide poisoning

One of the effects of organophosphate poisoning is inhibition of acetylcholinesterase leading to upregulation of nicotinic and muscarinic cholinergic neurotransmission. In warfare, sarin gas is a highly toxic synthetic organophosphorus compound that has similar effects.

Features can be predicted by the accumulation of acetylcholine (mnemonic = SLUD)

- Salivation
- Lacrimation
- Urination
- Defecation/diarrhoea
- cardiovascular: hypotension, bradycardia
- also: small pupils, muscle fasciculation

Management

- atropine
- the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 33 year female of Arabic descent with bullous pemphigus is admitted electively for intravenous immunoglobulins. She has no other past medical history, no recent travel history and has lived in rural Norfolk her whole life. On day 4 of a 5 day planned course of treatment, she develops pyrexia, nausea and vomiting. On examination, you note mild neck stiffness and photophobia. She also complains of a posterior headache. Her serum markers are as below:

WBC	14.0 * 10^9/l
Neuts	11.0 * 10^9/l

Na^+	133 mmol/l
K^+	3.8 mmol/l
Urea	4.7 mmol/l
Creatinine	80 µmol/l
CRP	4 mg/l

A CT head is unremarkable. The immunoglobulin is stopped. You perform a lumbar puncture: WCC 10/mm3, protein 0.4g/L, no organisms, opening pressure 17.8 cm H2O. Blood cultures taken during a temperature spike grow no organisms after 48 hours. What is the diagnosis?

- Aseptic meningitis
- Bullous pemphigus flare
- TB reactivation, resulting in TB meningitis
- Viral meningitis
- Bacterial meningitis

Submit answer

Reference ranges

Score: **15.1%**

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A CT head is unremarkable. The immunoglobulin is stopped. You perform a lumbar puncture: WCC 10/mm3, protein 0.4g/L, no organisms, opening pressure 17.8 cm H2O. Blood cultures taken during a temperature spike grow no organisms after 48 hours. What is the diagnosis?

Aseptic meningitis	79%
Bullous pemphigus flare	5%
TB reactivation, resulting in TB meningitis	9%
Viral meningitis	6%
Bacterial meningitis	1%

Aseptic meningitis is a classic complication of IVIg treatment. This patients inflammatory markers are unremarkable, as is the rest of her septic screen. There is little to suggest worsening of underlying bullous pemphigus. Aseptic meninigits occurs in 1% of patients with intravenous immunoglobulin. The key is to rule out any infective causes and to stop the immunoglobulin treatment until this is been done. Modern preparations can be stored in a fridge for months: there

does not need to be fears that expensive IVIg will be wasted if you dont continue with the treatment!

Discuss (8) Improve

Next question

Immunoglobulins: therapeutics

The Department of Health issued guidelines on the use of intravenous immunoglobulins in May 2008

Uses

- primary and secondary immunodeficiency
- idiopathic thrombocytopenic purpura
- myasthenia gravis
- Guillain-Barre syndrome
- Kawasaki disease
- toxic epidermal necrolysis
- pneumonitis induced by CMV following transplantation
- low serum IgG levels following haematopoietic stem cell transplant for malignancy
- dermatomyositis
- chronic inflammatory demyelinating polyradiculopathy

Basics

- formed from large pool of donors (e.g. 5,000)
- IgG molecules with a subclass distribution similar to that of normal blood
- half-life of 3 weeks

AI + Q 123

Next question

B I

Textbooks

High-yield textbook

Extended textbook

Links

Department of Health

1 1

2011 Guidelines on the clinical use of immunoglobulins

Suggest link Report broken link

Score: **18.1%**

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A 17-year-old girl presents to the emergency department with her mother. She is drowsy, ataxic, vomiting, her heart rate is 130 beats per minute and her blood pressure 130/84 mmHg. Her only past medical history is epilepsy which has recently been well controlled. After arguing with her boyfriend 40 minutes ago she took an unknown quantity of sodium valproate.

Following initial fluid resuscitation which of the following would you implement?

- Activated charcoal
- Observe for 3 hours and discharge
- Fomepizole
- Loperamide
- Transfer to psychiatric bed

Submit answer

Reference ranges

Score: **15.1%**

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Following initial fluid resuscitation which of the following would you implement?

Activated charcoal	86%
Observe for 3 hours and discharge	5%
Fomepizole	4%
Loperamide	1%
Transfer to psychiatric bed	4%

As the question states that she has taken the overdose less than an hour previously she would be suitable for activated charcoal.

Of the other options, discharging her while severely symptomatic, or transfer to a psychiatric ward while there are acute medical issues would be inappropriate.

Fomepizole is used in methanol or ethylene glycol poisoning.

The question does not give any indication for loperamide at present.

Discuss (4) | Improve

Next question

Overdose and poisoning: management

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization with IV bicarbonate • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil The majority of overdoses are managed with supportive care only due to the risk of seizures with flumazenil. It is generally only used with severe or iatrogenic overdoses.
Tricyclic antidepressants	Management • IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity • arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics
Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine

Toxin	Treatment
	• in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit
Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF

Emergency treatment of poisoning

2 1

Royal College of Physicians

2012 Poisoning and self-harm update

1 1

Royal College of Physicians

2007 Acute poisoning on the medical admissions unit

1 2

Suggest link

Report broken link

Score: **18.1%**

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You are called to review a 48-year-old woman in the Surgical Ward. She was admitted directly to the ward five hours ago post-operation for an elective open right hemicolectomy for adenocarcinoma of the caecum. The operation was completed without any concern. For postoperative pain relief, the patient has a local anaesthetic infusion to rectus sheath catheters and an oxycodone PCA. No significant previous medical history. No known drug allergies.

The patient had been reviewed by the nursing team half an hour before for postoperative antibiotics and replacement intravenous fluids. Her observations were all within normal parameters, however, the patient complained of new tinnitus.

She has just had a self-terminating seizure and remains post-ictal with a GCS of 10. Her observations are:

- Heart rate 45 bpm
- Blood pressure 78/35 mmHg
- Respiratory rate 25/min
- Temperature 36.5ºC
- Blood glucose 4.4mmol/l

What treatment should be started immediately?

- Intravenous levetiracetam loading dose
- Intravenous fluid and vasopressor
- Intravenous lipid emulsion
- Major haemorrhage transfusion
- Intravenous glucose

Submit answer

Reference ranges

Score: **15.1%**

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- Heart rate 45 bpm
- Blood pressure 78/35 mmHg
- Respiratory rate 25/min
- Temperature 36.5ºC
- Blood glucose 4.4mmol/l

What treatment should be started immediately?

Intravenous levetiracetam loading dose	2%
Intravenous fluid and vasopressor	14%
Intravenous lipid emulsion	81%
Major haemorrhage transfusion	1%
Intravenous glucose	2%

Local anaesthetic toxicity can present with CNS (e.g. reduced GCS, seizures) and cardiovascular (e.g. tachyarrhythmias, hypotension, bradyarrhythmias) features. Treatment of severe toxicity is with IV lipid emulsion.

Important for me Less important

Local anaesthetic toxicity is correct. The patient has a local anaesthetic infusion in place. This may be delivering local anaesthetic directly into the bloodstream or the local anaesthetic infusion may have inadvertently been connected to the intravenous access port. This is severe toxicity with seizures, bradycardia, hypotension and low GCS. Tinnitus, perioral numbness, metallic taste and auditory changes can be earlier symptoms. In this case of severe toxicity, lipid emulsion should be administered. This acts as a binding agent for the circulating local anaesthetic molecules.

Intravenous levetiracetam loading dose is incorrect. The patient's seizure is secondary to local anaesthetic toxicity and not epilepsy. Although further seizure control may be required the underlying cause should be treated immediately to prevent further seizures, cardiovascular collapse and death.

Intravenous fluid and vasopressor are incorrect because the patient's deteriorating clinical condition does not fit with sepsis. They have had an operation without complication, are already on prophylactic antibiotics and their clinical signs do not make sepsis the most likely diagnosis. IV fluids and vasopressors may be required in the course of their treatment for local anaesthetic toxicity but they do not treat the underlying cause of the deterioration.

Major haemorrhage transfusion is incorrect. Although postoperative haemorrhage should always be considered there is a more likely diagnosis in this case. Although the operation progressed well there is a risk of postoperative bleeding. If the patient is displaying symptoms of haemorrhage and cardiovascular shock then the surgical team and anaesthetic team must be notified immediately to facilitate an urgent return to theatre. Activation of the major haemorrhage protocol may be appropriate depending on the clinical picture.

Intravenous glucose is incorrect because the clinical scenario points towards local anaesthetic toxicity. Although it is important to ensure that glucose is correct to be within normal range, it is more important to start the lipid emulsion immediately. This will prevent further seizures, cardiovascular collapse and death.

Discuss (1) | Improve

Next question >

Local anaesthetic agents

Lidocaine

- An amide
- Local anaesthetic and a less commonly used antiarrhythmic (affects Na channels in the axon)
- Hepatic metabolism, protein bound, renally excreted

- Toxicity: due to IV or excess administration. Increased risk if liver dysfunction or low protein states. Note acidosis causes lidocaine to detach from protein binding. Local anesthetic toxicity can be treated with IV 20% lipid emulsion
- Drug interactions: Beta blockers, ciprofloxacin, phenytoin
- Features of toxicity: Initial CNS over activity then depression as lidocaine initially blocks inhibitory pathways then blocks both inhibitory and activating pathways. Cardiac arrhythmias.
- Increased doses may be used when combined with adrenaline to limit systemic absorption.

Cocaine

- Pure cocaine is a salt, usually cocaine hydrochloride. It is supplied for local anaesthetic purposes as a paste.
- It is supplied for clinical use in concentrations of 4 and 10%. It may be applied topically to the nasal mucosa. It has a rapid onset of action and has the additional advantage of causing marked vasoconstriction.
- It is lipophillic and will readily cross the blood brain barrier. Its systemic effects also include cardiac arrhythmias and tachycardia.
- Apart from its limited use in ENT surgery it is otherwise used rarely in mainstream surgical practice.

Bupivacaine

- Bupivacaine binds to the intracellular portion of sodium channels and blocks sodium influx into nerve cells, which prevents depolarization.
- It has a much longer duration of action than lignocaine and this is of use in that it may be used for topical wound infiltration at the conclusion of surgical procedures with long duration analgesic effect.
- It is cardiotoxic and is therefore contra indicated in regional blockage in case the tourniquet fails.
- Levobupivicaine (Chirocaine) is less cardiotoxic and causes less vasodilation.

Prilocaine

- Similar mechanism of action to other local anaesthetic agents. However, it is far less cardiotoxic and is therefore the agent of choice for intravenous regional anaesthesia e.g. Biers Block.

All local anaesthetic agents dissociate in tissues and this contributes to their therapeutic effect. The dissociation constant shifts in tissues that are acidic e.g. where an abscess is present, and this reduces the efficacy.

Doses of local anaesthetics

Agent	Dose plain	Dose with adrenaline
Lignocaine	3mg/Kg	7mg/Kg
Bupivacaine	2mg/Kg	2mg/Kg
Prilocaine	6mg/Kg	9mg/Kg

These are a guide only as actual doses depend on site of administration, tissue vascularity and co-morbidities.

Maximum total local anaesthetic doses

- Lignocaine 1% plain - 3mg/ Kg - 200mg (20ml)
- Lignocaine 1% with 1 in 200,000 adrenaline - 7mg/Kg - 500mg (50ml)
- Bupivicaine 0.5% - 2mg/kg- 150mg (30ml)

Maximum doses are based on ideal body weight

Effects of adrenaline

Adrenaline may be added to local anaesthetic drugs. It prolongs the duration of action at the site of injection and permits usage of higher doses (see above). It is contra indicated in patients taking MAOI's or tricyclic antidepressants. The toxicity of bupivacaine is related to protein binding and addition of adrenaline to this drug does not permit increases in the total dose of bupivacaine, in contrast to the situation with lignocaine.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Local anesthetics

Osmosis - YouTube 6 0

General & local anaesthetics

Speed Pharmacology - YouTube 2 0

Local anaesthetic toxicity

Kloss and Bruce - YouTube 4 1

Report broken media

Score: **18.1%**

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A 57-year-old man is bought to ED by ambulance after being found on the street unwell.

On examination, he is maintaining his own airway but is tachypnoeic with a respiratory rate of 28 breaths/minute. He complains of a headache and blurred vision and has poor coordination on examination.

After a short period in the department, he deteriorates further and is drowsy with a GCS of 11.

An arterial blood gas (ABG) shows:

pH	7.2	(7.35-7.45)
pO2	16.4 kPa	(10.0-13.0 kPa)
pCO2	2.3 kPa	(4.50-6.00 kPa)
HCO3-	14 mmol/L	(22.0-28.0 mmol/L)
BM	6.4 mmol/L	(3.9-8.0 mmol/L)
Na+	143 mmol/L	(135-145 mmol/L)
Cl-	98 mmol/L	(98-107 mmol/L)

What is the most appropriate next step in management for this patient?

- Activated charcoal
- Ethanol
- Fomepizole
- Haemodialysis
- Naloxone

Submit answer

Reference ranges

Score: **15.1%**

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BM	6.4 mmol/L	(3.9-8.0 mmol/L)
Na+	143 mmol/L	(135-145 mmol/L)
Cl-	98 mmol/L	(98-107 mmol/L)

What is the most appropriate next step in management for this patient?

Activated charcoal	3%
Ethanol	6%
Fomepizole	79%
Haemodialysis	10%
Naloxone	3%

Methanol poisoning is preferentially treated with IV fomepizole; this should be commenced immediately the suspicion is raised

Important for me Less important

This patient presents with symptoms of methanol poisoning (tachypnoea, headache, blurred vision, impaired coordination and reduced consciousness). The significant metabolic acidosis with raised anion gap should increase your suspicion. The immediate management of methanol poisoning is with the antidote **fomepizole**.

Activated charcoal has no effect to bind methanol and therefore plays no role in the use of methanol poisoning.

Ethanol can be used in methanol poisoning as it competitively binds to alcohol dehydrogenase and blocks methanol binding. However, it is no longer used first-line in the UK since the introduction of fomepizole.

Haemodialysis may be considered for this patient but fomepizole would be the next step in management while this was arranged.

Naloxone is used in opioid overdose but has no role in methanol poisoning.

Discuss (1) Improve

Next question

Methanol poisoning

Methanol poisoning causes both the effects associated with alcohol (intoxication, nausea etc) and also specific visual problems, including blindness. These effects are thought to be secondary to the accumulation of formic acid. The actual pathophysiology of methanol-associated visual loss is not fully understood but it is thought to be caused by a form of optic neuropathy

Management

- fomepizole (competitive inhibitor of alcohol dehydrogenase) or ethanol
- haemodialysis
- cofactor therapy with folinic acid to reduce ophthalmological complications

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

1 5

Suggest link

Report broken link

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A 65-year-old female presents to respiratory outpatients with an 8-week history of gradually worsening shortness of breath. Her exercise tolerance has reduced to 50 yards from several miles; she reports chronic lower back pain but no other symptoms. Her past medical history includes type 2 diabetes mellitus, psoriasis, recurrent urinary tract infections and ischaemic heart disease. She currently takes metformin 500 mg BD, bisoprolol 2.5 mg OD, aspirin 75 mg OD, atorvastatin 40 mg ON, ramipril 2.5 mg and nitrofurantoin 50 mg ON with no change in the last 6 months. She has never smoked, has no pets and works as a secretary. On examination, the patient is breathless on minimal exertion. Observations are normal, there is no clubbing and she has fine end-inspiratory crepitations at both bases. A CT chest shows ground glass changes with minimal honey combing at both bases. What is the most likely cause of her underlying respiratory condition?

- Asbestosis
- Psoriasis
- Metformin
- Ankylosing spondylitis
- Nitrofurantoin

Submit answer

Reference ranges

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Asbestosis	4%
Psoriasis	2%
Metformin	1%
Ankylosing spondylitis	6%
Nitrofurantoin	**87%**

This patient has a history and examination consistent with pulmonary fibrosis. Her CT chest supports this demonstrating ground glass and honey combing at both bases. Pulmonary function tests will demonstrate a restrictive pattern and arterial blood gas analysis will show a type 1 respiratory failure. Long-term use of nitrofurantoin is known to cause a basal pattern of pulmonary fibrosis and is the most likely culprit in this case. There is no suggestion of asbestos exposure in the history and both psoriasis and ankylosing spondylitis classically cause an apical pattern of fibrosis. Lower back pain is a common medical complaint in this age group and ankylosing spondylitis tends to present in younger males.

Discuss (3) | Improve

Next question

Drugs causing lung fibrosis

Causes

- amiodarone
- cytotoxic agents: busulphan, bleomycin
- anti-rheumatoid drugs: methotrexate, sulfasalazine
- nitrofurantoin
- ergot-derived dopamine receptor agonists (bromocriptine, cabergoline, pergolide)

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 64-year-old woman comes to the Emergency department complaining of palpitations at rest, and worsening angina over the past month. She has been treated with amiodarone for the past 3 years for recurrent ventricular tachycardia. She has a blood pressure of 110/70 mmHg, pulse of 95 beats per minute, and a fine tremor. There is no goitre. TSH is suppressed at less than 0.05 U/ml.

You suspect she has amiodarone induced thyrotoxicosis. How best can you determine the underlying pathophysiology?

- IL6 level
- Thyroglobulin level
- TSH level
- Duration of amiodarone therapy
- Colour flow doppler ultrasonography

Submit answer

Reference ranges

Score: **15.1%**

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You suspect she has amiodarone induced thyrotoxicosis. How best can you determine the underlying pathophysiology?

IL6 level	12%
Thyroglobulin level	24%
TSH level	7%
Duration of amiodarone therapy	5%
Colour flow doppler ultrasonography	52%

Type 1 amiodarone induced thyrotoxicosis is caused by increased production of thyroid hormone, most likely as a result of the excess iodine load administered to the patient as a result of amiodarone treatment. Type 2 amiodarone induced thyrotoxicosis is caused by a destructive thyroiditis. Colour flow doppler ultrasonography needs to be performed by an experienced operator, but is thought to distinguish between the two causes of amiodarone induced thyrotoxicosis around 80% of the time. In patients where the diagnosis is uncertain, radioiodine uptake, (which is low in type 2 disease) my further help differentiating between the two.

Type 2 disease is generally seen later in the time course of amiodarone therapy, although this isn't an invariable differentiating factor, and data on the usefulness of IL6 and thyroglobulin is conflicting.

Reference on use of colour flow doppler:

http://www.ncbi.nlm.nih.gov/pubmed/11849244

Next question

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

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Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

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An 82-year-old man presents with pain following a fall. He was brought in by ambulance after he slipped and hurt himself on the pavement. He ended up tripping whilst returning from the local shops. He remembers his fall and noted immediate pain in his right leg, and was unable to get up. A passerby called an ambulance and he was brought into the hospital. He has a past medical history of ischaemic heart disease, mild dementia, hypertension and high cholesterol.

Pelvic X-rays demonstrated an intertrochanteric right hip fracture. He is due to be operated on within the five hours and is put nil by mouth and started on IV fluids. How should his risk of venous thromboembolism (VTE) be managed?

- Low molecular weight heparin before surgery
- Unfractionated heparin before surgery
- Delay VTE prophylaxis until following surgery
- Mechanical VTE prophylaxis before surgery
- IV heparin infusion until surgery

Submit answer

Reference ranges

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An 82-year-old man presents with pain following a fall. He was brought in by ambulance after he slipped and hurt himself on the pavement. He ended up tripping whilst returning from the local shops. He remembers his fall and noted immediate pain in his right leg, and was unable to get up. A passerby called an ambulance and he was brought into the hospital. He has a past medical history of ischaemic heart disease, mild dementia, hypertension and high cholesterol.

Pelvic X-rays demonstrated an intertrochanteric right hip fracture. He is due to be operated on within the five hours and is put nil by mouth and started on IV fluids. How should his risk of venous thromboembolism (VTE) be managed?

Low molecular weight heparin before surgery	21%
Unfractionated heparin before surgery	17%
Delay VTE prophylaxis until following surgery	23%
Mechanical VTE prophylaxis before surgery	31%
IV heparin infusion until surgery	8%

The correct answer is mechanical VTE prophylaxis before surgery. This is a patient with a hip fracture due to be operated on within 12 hours. He should have careful mechanical VTE prophylaxis with stockings or pneumatic compression despite having a fracture. If surgery was to occur in over 12 hours then low molecular weight heparin should be started, with the last dose given 12 hours before surgery. If he also had renal failure, then unfractionated heparin should be used and also stopped 12 hours before surgery.

Source:

'Venous thromboembolism: reducing the risk for patients in hospital' Clinical guideline [CG92]. The National Institute for Health and Care Excellence, January 2010.

Heparin ★

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia (HIT) Osteoporosis	Bleeding Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin

- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
    - direct thrombin inhibitor e.g. argatroban
    - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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A 56-year-old man is brought into hospital with palpitations, restlessness and agitation. His wife tells you he has lost 2 kilograms of weight over the past month. She attributes this to persistent diarrhoea over the same period.

He has a past medical history of persistent atrial fibrillation and takes amiodarone.

On examination, he has a heart rate of 144 beats per minute and his ECG confirms atrial fibrillation with no ischaemic changes. You are unable to palpate any abnormality of the thyroid cartilage.

Investigations are as follows:

TSH	0.1 mU/L
^{99}Tc scan	reduced uptake

Which of the following is the most important step in this patient's immediate management?

- Carbimazole
- Corticosteroids
- Propylthiouracil
- Thyroidectomy
- Increase dose of amiodarone

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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He has a past medical history of persistent atrial fibrillation and takes amiodarone.

On examination, he has a heart rate of 144 beats per minute and his ECG confirms atrial fibrillation with no ischaemic changes. You are unable to palpate any abnormality of the thyroid cartilage.

Investigations are as follows:

TSH	0.1 mU/L
^{99}Tc scan	reduced uptake

Which of the following is the most important step in this patient's immediate management?

Carbimazole	9%
Corticosteroids	83%
Propylthiouracil	6%
Thyroidectomy	1%
Increase dose of amiodarone	1%

In this case, amiodarone is likely the underlying cause of this man's fast AF. Indeed he has a number of symptoms suggestive of hyperthyroidism.

The mainstay of treatment, in this case, would be to give steroids. The absence of a goitre points towards type 2 AIT. The reduced uptake on radioisotope imaging reflects the destructive changes to the thyroid seen in this type of AIT.

Increasing the dose of amiodarone may help with cardioversion but will not treat the underlying cause. Treating with anti-thyroid drugs, such as carbimazole or PTU, may help with thyrotoxicosis but again will not reverse the destructive changes to the thyroid gland. At this stage surgical removal of the thyroid is not indicated.

Amiodarone and the thyroid gland

Around 1 in 6 patients taking amiodarone develop thyroid dysfunction

Amiodarone-induced hypothyroidism

The pathophysiology of amiodarone-induced hypothyroidism (AIH) is thought to be due to the high iodine content of amiodarone causing a Wolff-Chaikoff effect*

Amiodarone may be continued if this is desirable

Amiodarone-induced thyrotoxicosis

Amiodarone-induced thyrotoxicosis (AIT) may be divided into two types:

	AIT type 1	**AIT type 2**
Pathophysiology	Excess iodine-induced thyroid hormone synthesis	Amiodarone-related destructive thyroiditis
Goitre	Present	Absent
Management	Carbimazole or potassium perchlorate	Corticosteroids

Unlike in AIH, amiodarone should be stopped if possible in patients who develop AIT

*an autoregulatory phenomenon where thyroxine formation is inhibited due to high levels of circulating iodide

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NCBI

5 3

Amiodarone-induced thyrotoxicosis: type 1 or type 2?

Suggest link Report broken link

Score: **18.1%**

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You are asked to review a 23-year-old patient with a history of ulcerative colitis who was admitted one week ago with a flare unresponsive to steroids. However, over the last three days, he has noticed a visible tremor, and the nurses have noted higher blood sugars on the ward.

On examination, his BP is 154/104mmHg, and his fasting blood glucose is 6.0. His abdomen is soft, with generalised lower abdominal discomfort but no guarding. His chest is clear.

He reports feeling more stressed recently at work and tells you he often uses cannabis in his downtime. The nurses have noticed him leaving the ward often during their shift.

Which medication was he most likely started on?

- Azathioprine
- Ciclosporin
- Infliximab
- Mesalazine
- Prednisolone

Submit answer

Reference ranges

Score: **15.1%**

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On examination, his BP is 154/104mmHg, and his fasting blood glucose is 6.0. His abdomen is soft, with generalised lower abdominal discomfort but no guarding. His chest is clear.

He reports feeling more stressed recently at work and tells you he often uses cannabis in his downtime. The nurses have noticed him leaving the ward often during their shift.

Which medication was he most likely started on?

Azathioprine	14%
Ciclosporin	50%
Infliximab	8%
Mesalazine	12%
Prednisolone	16%

Cannabidiol may increase the concentration of ciclosporin

Important for me Less important

Ciclosporin is the correct answer. In this case, a patient with a severe flare of UC refractory to steroids might be started on ciclosporin. This question assesses knowledge of ciclosporin's side effects and its interaction with cannabis. Ciclosporin may interact with cannabidiol, and adverse effects include tremor, impaired glucose tolerance and susceptibility to infection. Cannabis use can increase ciclosporin levels due to inhibition of cytochrome P450 enzymes by cannabinoids such as CBD.

The other medications listed are used in the treatment of ulcerative colitis but do not exhibit this combination of side effects or interactions with cannabis (as seen on the BNF interaction checker).

Azathioprine commonly interacts with cannabidiol. The levels of azathioprine are increased as cannabidiol decreases its metabolism. Side effects of azathioprine include bone marrow

suppression, thrombocytopenia and increased susceptibility to infections. However, the mentioned side effects in the case don't align with azathioprine side effects.

Infliximab is not known to interact with cannabidiol. Side effects include diarrhoea, infusion-related reactions and increased susceptibility to infections.

Mesalazine is not known to interact with cannabidiol. Side effects include arthralgia, pancreatitis (rarely), skin reactions and diarrhoea.

Prednisolone is not known to interact with cannabidiol. It is unlikely that this patient would be on this medication if the current episode is unresponsive to steroids. Side effects include fluid retention, increased risk of infection, peptic ulcer and hypertension.

Discuss | Improve

Next question >

Ciclosporin ★

Ciclosporin is an immunosuppressant which decreases clonal proliferation of T cells by reducing IL-2 release. It acts by binding to cyclophilin forming a complex which inhibits calcineurin, a phosphatase that activates various transcription factors in T cells

Adverse effects of ciclosporin (note how everything is increased - fluid, BP, K^+, hair, gums, glucose)

- nephrotoxicity
- hepatotoxicity
- fluid retention
- hypertension
- hyperkalaemia
- hypertrichosis
- gingival hyperplasia
- tremor
- impaired glucose tolerance
- hyperlipidaemia
- increased susceptibility to severe infection

Interestingly for an immunosuppressant, ciclosporin is noted by the BNF to be 'virtually non-myelotoxic'.

Notable interactions

- cannabidiol: may increase the concentration of ciclosporin BNF

Indications

- following organ transplantation
- rheumatoid arthritis
- psoriasis (has a direct effect on keratinocytes as well as modulating T cell function)
- ulcerative colitis
- pure red cell aplasia

Next question

Textbooks

High-yield textbook

Extended textbook

Links

BNF 2 1

Ciclosporin

British Association of Dermatologists 1 1

2018 Guidelines for the safe and effective prescribing of oral ciclosporin in dermatolog

Suggest link Report broken link

Score: **18.1%**

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A 45 year-old Russian woman complains of a 2 week history of joint pain affecting her wrists, shoulders and knees bilaterally. She also reports calf pain bilaterally. She has been started on first line treatment for pulmonary tuberculosis 2 months previously. Symptoms resolved after the addition of aspirin.

Which drug is most likely causing her symptoms?

- Rifampicin
- Isoniazid
- Pyrazinamide
- Ethambutol
- Streptomycin

Submit answer

Reference ranges

Score: **15.1%**

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Which drug is most likely causing her symptoms?

Rifampicin	7%
Isoniazid	22%
Pyrazinamide	62%
Ethambutol	3%
Streptomycin	6%

By far the most common cause of arthralgia from the above is pyrazinamide.

Discuss (2) | Improve

Next question

Tuberculosis: drug side-effects and mechanism of action

Rifampicin

- mechanism of action: inhibits bacterial DNA dependent RNA polymerase preventing transcription of DNA into mRNA
- potent liver enzyme inducer
- hepatitis, orange secretions
- flu-like symptoms

Isoniazid

- mechanism of action: inhibits mycolic acid synthesis

- peripheral neuropathy: prevent with pyridoxine (Vitamin B6)
- hepatitis, agranulocytosis
- liver enzyme inhibitor

Pyrazinamide

- mechanism of action: converted by pyrazinamidase into pyrazinoic acid which in turn inhibits fatty acid synthase (FAS) I
- hyperuricaemia causing gout
- arthralgia, myalgia
- hepatitis

Ethambutol

- mechanism of action: inhibits the enzyme arabinosyl transferase which polymerizes arabinose into arabinan
- optic neuritis: check visual acuity before and during treatment
- dose needs adjusting in patients with renal impairment

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

NICE

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2016 Tuberculosis guidelines

Suggest link Report broken link

Media

Tuberculosis

Osmosis - YouTube 0 0

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Score: **18.1%**

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A 32-year-old male illicit drug user is caught attempting to sell cocaine on the street by the police. On seeing the officers approach him, he swallows a plastic bag containing around 1.5g of cocaine in an attempt to destroy the evidence. The police officers bring the gentleman into the emergency department. He is asymptomatic and complains of no chest pain. His ECG is unremarkable and his admission blood tests demonstrate no abnormalities, including a negative troponin. What should you do next?

- Treat presumptively as acute coronary syndrome
- Discharge the patient to police custody
- Await 12 hour troponin and if negative, discharge to police custody
- Repeat ECG and if no dynamic changes, discharge to police custody
- Monitor the patient until the ingested cocaine is excreted

Submit answer

Reference ranges

Score: **15.1%**

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A 32-year-old male illicit drug user is caught attempting to sell cocaine on the street by the police. On seeing the officers approach him, he swallows a plastic bag containing around 1.5g of cocaine in an attempt to destroy the evidence. The police officers bring the gentleman into the emergency department. He is asymptomatic and complains of no chest pain. His ECG is unremarkable and his admission blood tests demonstrate no abnormalities, including a negative troponin. What should you do next?

Treat presumptively as acute coronary syndrome	3%
Discharge the patient to police custody	3%
Await 12 hour troponin and if negative, discharge to police custody	14%
Repeat ECG and if no dynamic changes, discharge to police custody	5%
Monitor the patient until the ingested cocaine is excreted	75%

Cocaine produces significant cardiovascular sequelae, classically coronary vasospasm resulting in cardiac ischaemia, presenting as chest pain and similar ECG changes to acute coronary syndrome. Similar vasospasm can lead to transient cerebral ischaemia, resulting in ischaemic infarcts. In addition, cardiac dysrhythmias are also common, including sinus tachycardia, sinus bradycardia, supraventricular tachyarrhythmias, bundle branch block, ventricular fibrillation and torsades de pointes. In this scenario, it is clear that the ingested cocaine has not penetrated to coverings and released systemically. However, the danger remains until the ingested cocaine has been excreted. Patients like this should be monitored for cardiovascular and cerebral sequelae until the ingested amounts are collected.

1. Egred M, Davis GK. Cocaine and the heart. Postgrad Med J 2005; 81: 568-71

Discuss (7) | Improve

Next question >

Cocaine

Cocaine is an alkaloid derived from the coca plant. It is widely used as a recreational stimulant. The price of cocaine has fallen sharply in the past decade resulting in cocaine toxicity becoming a much more frequent clinical problem. This increase has made cocaine a favourite topic of question writers.

Mechanism of action

- cocaine blocks the uptake of dopamine, noradrenaline and serotonin

Adverse effects

- cardiovascular
  - coronary artery spasm → myocardial ischaemia/infarction
  - both tachycardia and bradycardia may occur
  - hypertension
  - QRS widening and QT prolongation
  - aortic dissection
- neurological
  - seizures
  - mydriasis
  - hypertonia
  - hyperreflexia
- psychiatric effects
  - agitation
  - psychosis
  - hallucinations
- others
  - ischaemic colitis is recognised in patients following cocaine ingestion. This should be considered if patients complain of abdominal pain or rectal bleeding
  - hyperthermia
  - metabolic acidosis
  - rhabdomyolysis

Management of cocaine toxicity

- in general, benzodiazepines are generally first-line for most cocaine-related problems
- chest pain:
  - benzodiazepines + glyceryl trinitrate
  - if myocardial infarction develops then primary percutaneous coronary intervention
- hypertension: benzodiazepines + sodium nitroprusside
- the use of beta-blockers in cocaine-induced cardiovascular problems is a controversial issue
  - the American Heart Association issued a statement in 2008 warning against the use of beta-blockers (due to the risk of unopposed alpha-mediated coronary vasospasm) but many cardiologists since have questioned whether this is valid
  - if a reasonable alternative is given in an exam it is probably wise to choose it

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Cocaine Dependence

Osmosis - YouTube 4 2

Report broken media

Score: **18.1%**

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Back to top

You are asked to review a 44-year-old lady in the emergency department. She is well known to the mental health liaison team because of her multiple suicide attempts. She is agitated and slightly confused and unable to give a precise history. She is able to tell you that she can't see clearly and has a dry mouth.

Her sister explains that the patient has been prescribed medicines for chronic neuropathic back pain and has taken them all after splitting up with her boyfriend.

On examination she has symmetrically dilated pupils but no focal motor deficit. Her heart sounds are normal and her lung fields are clear on auscultation. An ECG shows sinus tachycardia with a QRS of 170.

Bloods results are as follows:

Hb	125 g/l
Platelets	440 * 10^9/l
WBC	9.9 * 10^9/l
Na^+	135 mmol/l
K^+	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	88 µmol/l
Plasma Paracetamol	<5mmol/l

You request an arterial blood gas which shows the following readings:

pH	7.29
pCO_2	3.4 kPa
pO_2	14.5 kPa

What is the most appropriate next step in this patient's management?

- IV sodium bicarbonate

- Haemodialysis
- IV lignocaine
- IV amiodarone
- Naloxone

Submit answer

Reference ranges

Score: **15.1%**

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You request an arterial blood gas which shows the following readings:

pH	7.29
pCO_2	3.4 kPa
pO_2	14.5 kPa

What is the most appropriate next step in this patient's management?

IV sodium bicarbonate 91%

Haemodialysis	4%
IV lignocaine	1%
IV amiodarone	1%
Naloxone	3%

The culprit here is a tricyclic antidepressant, such as dosulepin or amitriptyline. The first step in treating this patient is to reverse the metabolic acidosis with IV bicarbonate. Dialysis is of no benefit in this scenario.

The dilated pupils and absence of respiratory depression point away from opioid overdose, making naloxone the wrong answer.

Discuss (3) Improve

Next question

Tricyclic overdose

Overdose of tricyclic antidepressants is a common presentation to emergency departments. Amitriptyline and dosulepin (dothiepin) are particularly dangerous in overdose.

Early features relate to anticholinergic properties: dry mouth, dilated pupils, agitation, sinus tachycardia, blurred vision.

Features of severe poisoning include:

- arrhythmias
- seizures
- metabolic acidosis
- coma

ECG changes include:

- sinus tachycardia
- widening of QRS
- prolongation of QT interval

Widening of QRS > 100ms is associated with an increased risk of seizures whilst QRS > 160ms is

associated with ventricular arrhythmias

Management

- IV bicarbonate
  - first-line therapy for hypotension or arrhythmias
  - indications include widening of the QRS interval >100 msec or a ventricular arrhythmia
- other drugs for arrhythmias

+ class 1a (e.g. quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation

- class III drugs such as amiodarone should also be avoided as they prolong the QT interval
- response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in the management of tricyclic-induced arrhythmias

- intravenous lipid emulsion is increasingly used to bind free drug and reduce toxicity
- dialysis is ineffective in removing tricyclics

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 56 year old gardener is brought to the Emergency Department by his wife. He was bitten on the right hand by a snake while clearing bushes in his back garden. It is identified as a common European adder. There is an obvious, red puncture site on the dorsum of the right hand with significant swelling of the entire hand to the level of the wrist. He is in significant discomfort. Examination reveals a normal cardiovascular examination with a heart rate of 112bpm, blood pressure of 137/90mmHg and normal respiratory rate and oxygen saturations. Capillary glucose is 5.6mmol. ECG shows normal sinus rhythm with normal PR, QRS and QT intervals. Routine blood tests including liver function, electrolytes, creatine kinase, full blood count and arterial blood gases are normal.

Which of the following interventions is the most appropriate at this stage?

- Application of a tourniquet above the site of envenomation to prevent systemic toxicity
- Bathe the site of envenomation in hot, sterile water to inactivate the venom
- Intravenous access and rapidly infuse 0.9% sodium chloride with 1g intravenous paracetamol
- Intravenous access and administer 200mg IV hydrocortisone, 10mg IV chlorphenamine and 0.5mg IM adrenaline 1:1000
- Intravenous access and administer 10ml adder antivenin in 500ml 0.9% sodium chloride over 30 minutes

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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Which of the following interventions is the most appropriate at this stage?

Application of a tourniquet above the site of envenomation to prevent systemic toxicity	13%
Bathe the site of envenomation in hot, sterile water to inactivate the venom	8%
Intravenous access and rapidly infuse 0.9% sodium chloride with 1g intravenous paracetamol	13%
Intravenous access and administer 200mg IV hydrocortisone, 10mg IV chlorphenamine and 0.5mg IM adrenaline 1:1000	7%
Intravenous access and administer 10ml adder antivenin in 500ml 0.9% sodium chloride over 30 minutes	58%

Adder bites are rare, but when they occur may be extremely painful; the mainstay of treatment is analgesia and supportive therapy. Discuss the use of antivenin with NPIS and do not apply a tourniquet

Important for me Less important

Application of a tourniquet is absolutely contraindicated since it increases the likelihood of localised necrosis of the affected limb as well as the risk of venous thrombosis which in turn increases the likelihood of central venous thrombosis on release of the tourniquet, as well as cardiac arrhythmia due to the systemic flooding of released potassium and other necrosis factors from tissue damage and haemolysis in a ligatured extremity when the tourniquet is removed.

Unlike venom of the United Kingdom's only other native poisonous animal, the weeverfish

(Trachinidae family), viper venom is not heat labile. Application of cold water to a bite may help to assuage pain, but hot water will not deactivate the venom. Weeverfish stings may be treated by bathing the affected limb (usually a foot) in hot water to denature the active proteins.

Adder antivenin is readily available for use in severe bites and can be obtained by contacting local zoos or the National Poisons Information Service. However, severe anaphylactic reactions to the antivenin are common and may be seen in up to 20% of recipients, particularly in atopic or asthmatic individuals. For this reason, antivenin is reserved only for severe systemic envenomations in patients with resistant hypotension, new ECG changes, significant rise in white cell count, raised CK, metabolic acidosis or swelling involving more than half the affected limb or crossing a joint boundary, e.g. beyond the wrist if bitten on the hand. The patient in the above scenario has none of these symptoms. Similarly, in the absence of any symptoms suggestive of an acute anaphylactoid reaction, treatment with adrenaline and hydrocortisone is unnecessary, although antihistamines may help symptomatically. Often, symptomatic treatment is all that is required with analgesia taking priority. Intravenous analgesia is often faster acting and fluids may be required if swelling is significant or vomiting is profuse.

Discuss (14) Improve

Next question ›

Snake bites in the UK ★

The common European adder or viper, vipera berus, is the only poisonous snake in the United Kingdom. Adder bites are relatively rare, even in rural areas, and often victims of snake bites are children. Even when bites occur, actual envenomation may not happen. Fatalities are extremely uncommon and since 1876 there have only been 14 recorded deaths due to adder envenomation. Bites by snakes and venom toxicity may be extremely painful locally however, and systemic symptoms may be present. Typical symptoms which may be expected following an adder envenomation include severe local pain, swelling, erythema, paraesthesia, numbness and blood blistering. In the ensuing hours, tracking erythema and significant swelling, often along the lymphatic system, may be seen, as well as compartment syndrome and tissue necrosis. Systemic symptoms similar to anaphylaxis may be seen with hypotension, collapse, airways swelling and compromise, diarrhoea, vomiting and fever; sometimes these may be delayed symptoms. Significant bruising at the site of skin puncture may occur due to the natural pro-coagulants within the venom. Other symptoms are due to the composition of the venom including cytokines, histamine and myriad enzymes.

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 25-year-old man attends the emergency department with his partner. He reports having taken an overdose of paracetamol approximately 30 hours ago. Initially, he vomited several times but has not done so for the last 10 hours. He complains of mild vague abdominal discomfort.

On examination, he has some right upper quadrant tenderness on palpation. There is no guarding and there is no obvious jaundice present.

What is the most appropriate management?

- Send paracetamol levels and await the result to decide on N-acetylcysteine
- Start N-acetylcysteine after taking paracetamol levels
- Start N-acetylcysteine and then take paracetamol levels
- Start N-acetylcysteine, paracetamol levels not required
- Supportive management

Submit answer

Reference ranges

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On examination, he has some right upper quadrant tenderness on palpation. There is no guarding and there is no obvious jaundice present.

What is the most appropriate management?

Send paracetamol levels and await the result to decide on N-acetylcysteine	13%
Start N-acetylcysteine after taking paracetamol levels	39%
Start N-acetylcysteine and then take paracetamol levels	22%
Start N-acetylcysteine, paracetamol levels not required	18%
Supportive management	7%

Paracetamol overdose: if presentation > 24 hours after an overdose start acetylcysteine if the patient is jaundiced, has hepatic tenderness or an elevated ALT

Important for me Less important

For patients who have taken an overdose of paracetamol with presentation later than 24 hours, start treatment before blood tests are available if the patient has visible jaundice or hepatic tenderness or has taken 150mg/kg or more of paracetamol as per Toxbase guidance, and start treatment after blood results if there is a raised ALT or INR, or detectable paracetamol level.

Given the clinical picture, this patient's right upper quadrant tenderness is highly likely to represent hepatic tenderness so he should be commenced on treatment. The best practice is to take the paracetamol levels before starting the infusion. Hence the option to **start N-acetylcysteine after taking paracetamol levels** is correct. This is because if paracetamol levels are taken during N-acetylcysteine infusion, some analysers may significantly underestimate paracetamol levels.

As this patient has hepatic tenderness, it would not be correct to wait for the levels before starting treatment. Hence, the option to **send paracetamol levels and await the result to decide on N-**

acetylcysteine is incorrect.

Due to this potential interference with the analyser, the option to **start N-acetylcysteine and then take paracetamol levels** is incorrect.

Taking paracetamol levels is helpful in management and is recommended by Toxbase. Therefore, the option to **start N-acetylcysteine, paracetamol levels not required** is incorrect.

This patient warrants N-acetylcysteine treatment, therefore **supportive management** is incorrect.

Discuss | Improve

Next question >

Paracetamol overdose: management ★

The following is based on 2012 Commission on Human Medicines (CHM) review of paracetamol overdose management. The big change in these guidelines was the removal of the 'high-risk' treatment line on the normogram. All patients are therefore treated the same regardless of risk factors for hepatotoxicity. The National Poisons Information Service/TOXBASE should always be consulted for situations outside of the normal parameters.

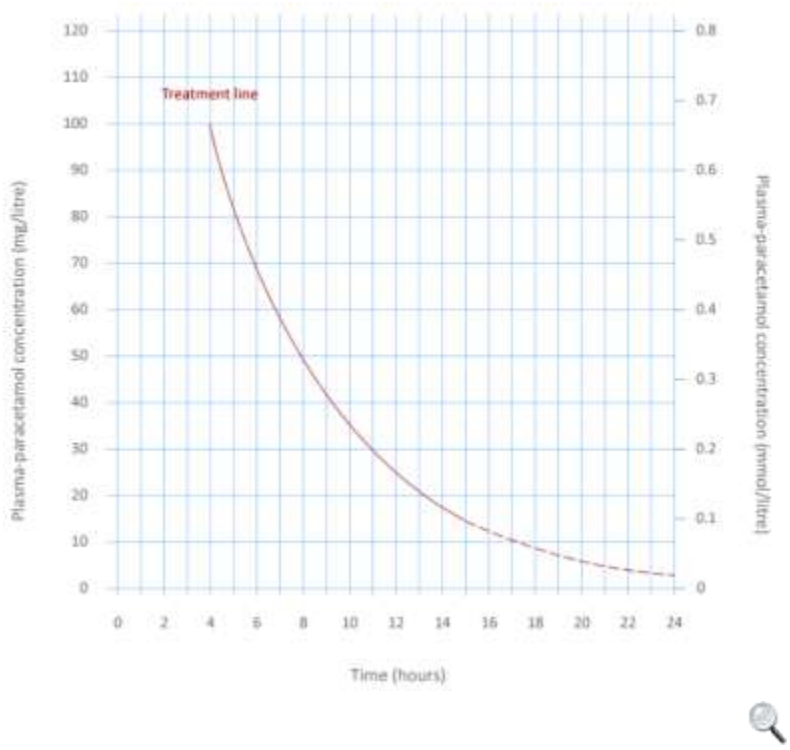
The minority of patients who present within 1 hour may benefit from activated charcoal to reduce absorption of the drug.

Acetylcysteine should be given if:

- the plasma paracetamol concentration is on or above a single treatment line joining points of 100 mg/L at 4 hours and 15 mg/L at 15 hours, regardless of risk factors of hepatotoxicity
- there is a staggered overdose* or there is doubt over the time of paracetamol ingestion, regardless of the plasma paracetamol concentration; or
- patients who present 8-24 hours after ingestion of an acute overdose of more than 150 mg/kg of paracetamol even if the plasma-paracetamol concentration is not yet available
- patients who present > 24 hours if they are clearly jaundiced or have hepatic tenderness, their ALT is above the upper limit of normal
    - acetylcysteine should be continued if the paracetamol concentration or ALT remains elevated whilst seeking specialist advice

Acetylcysteine is now infused over 1 hour (rather than the previous 15 minutes) to reduce the number of adverse effects. Acetylcysteine commonly causes an anaphylactoid reaction (non-IgE mediated mast cell release). Anaphylactoid reactions to IV acetylcysteine are generally treated by

stopping the infusion, then restarting at a slower rate.



King's College Hospital criteria for liver transplantation (paracetamol liver failure)

Arterial pH < 7.3, 24 hours after ingestion

or all of the following:

- **prothrombin time > 100 seconds**
- **creatinine > 300 µmol/l**
- **grade III or IV encephalopathy**

*an overdose is considered staggered if all the tablets were not taken within 1 hour

AI + Q 123

Next question >

B I A

Textbooks

High-yield textbook

Extended textbook

Links

Medicines and Healthcare products Regulatory Agency

3 3

2012 Paracetamol overdose update

Suggest link Report broken link

Score: **18.1%**

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Blood results are as follows:

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	85 * 10^9/L	(150 - 400)
WBC	20.8 * 10^9/L	(4.0 - 11.0)
Na^+	138 mmol/L	(135 - 145)
K^+	3.5 mmol/L	(3.5 - 5.0)
Urea	8.4 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Glucose	5.1 mmol/L	(3.9 - 5.6)
Serum osmolarity	310 mOsm/kg	(275 - 295)

What is the treatment of choice?

- Activated charcoal
- Ethanol
- Folinic acid
- Fomepizole
- Haemodialysis

Submit answer

Reference ranges

Score: **15.1%**

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Creatinine	110 µmol/L	(55 - 120)
CRP	22 mg/L	(< 5)
Glucose	5.1 mmol/L	(3.9 - 5.6)
Serum osmolarity	310 mOsm/kg	(275 - 295)

What is the treatment of choice?

Activated charcoal	2%
Ethanol	7%
Folinic acid	10%
Fomepizole	77%
Haemodialysis	5%

Ethylene glycol toxicity management - fomepizole. Also ethanol / haemodialysis

Important for me Less important

The patient has clinical and biochemical features of ethylene glycol toxicity.

The osmolar gap = osmolality (measured) - osmolarity (calculated). A normal osmolar gap should be < 10.

The calculated osmolarity in this case = (2 x [Na+]) + [glucose] + [urea]) = 289.5 mOsm/kg.

Therefore the osmolar gap = 310 - 289.5 = 20.5 mOsm/kg.

The patient thus has a high osmolar gap which indicates the presence of an abnormal solute present in significant amounts.

Causes of a high osmolar gap include:

- Mannitol
- Methanol
- Ethylene glycol

Fomepizole is correct. For patients with ethylene glycol toxicity, early administration of fomepizole or ethanol will minimise the further metabolism of ethylene glycol and the development of clinical and metabolic complications. Fomepizole is the agent of choice if available.

Activated charcoal is incorrect. Activated charcoal is of no use since it does not adsorb significant quantities of ethylene glycol.

Ethanol is incorrect. Fomepizole is the preferred antidote since, unlike ethanol, it does not require regular monitoring of blood concentrations or cause inebriation. Ethanol is an effective antidote but is associated with more complications. If there is any delay in obtaining fomepizole, administer ethanol urgently initially, followed by fomepizole when available.

Folinic acid is incorrect. Folinic acid is used to reduce ophthalmological complications in methanol poisoning.

Haemodialysis is incorrect. The patient is symptomatic and requires urgent treatment. Haemodialysis has a role in severe or refractory cases.

Discuss (3) | Improve

Next question >

Ethylene glycol toxicity ★

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

4 1

Toxic Alcohol Ingestion

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A 20-year-old university student attends the emergency department with his friends who were concerned after they found him urinating on the living room sofa. On examination, he appeared agitated, sweaty, and had occasional muscle twitching.

Observations: heart rate 110 beats per minute, blood pressure 165/105 mmHg, temperature 37.9ºC.

On review of his medical record, you can see he takes citalopram for depression and has also recently been started on another medication.

What other medication is most likely to account for his presentation?

- Cyclizine
- Cyproheptadine
- Methylene blue
- Phenoxymethylpenicillin
- Tramadol

Submit answer

Reference ranges

Score: **15.1%**

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On review of his medical record, you can see he takes citalopram for depression and has also recently been started on another medication.

What other medication is most likely to account for his presentation?

Cyclizine	11%
Cyproheptadine	14%
Methylene blue	4%
Phenoxymethylpenicillin	3%
Tramadol	**69%**

Tramadol co-prescribed with SSRIs is a common cause of serotonin syndrome

Important for me Less important

The patient in the stem is likely presenting with serotonin syndrome, as indicated by disorientation, agitation, muscle twitching, hypertension, tachycardia, and pyrexia.

Tramadol is the correct answer. When this is co-prescribed with SSRIs such as citalopram it can precipitate serotonin syndrome. This is because tramadol acts as a central serotonin reuptake inhibitor and so further increases serotonin levels.

Cyclizine is incorrect. There is no evidence to suggest that cyclizine is implicated in the development of serotonin syndrome.

Cyproheptadine is incorrect. Cyproheptadine can be used as a treatment for serotonin syndrome. Other treatments can include intravenous fluids and benzodiazepines.

Methylene blue is incorrect. It is a thiazine dye used to treat methemoglobinemia and is not implicated in the development of serotonin syndrome.

Phenoxymethylpenicillin is incorrect. Other antibiotics such as ciprofloxacin, isoniazid, and linezolid can potentiate serotonin syndrome, however, there is no evidence to suggest that phenoxymethylpenicillin is a cause.

👍 👎 💬 Discuss (1) Improve

Next question ›

Serotonin syndrome ★

Causes

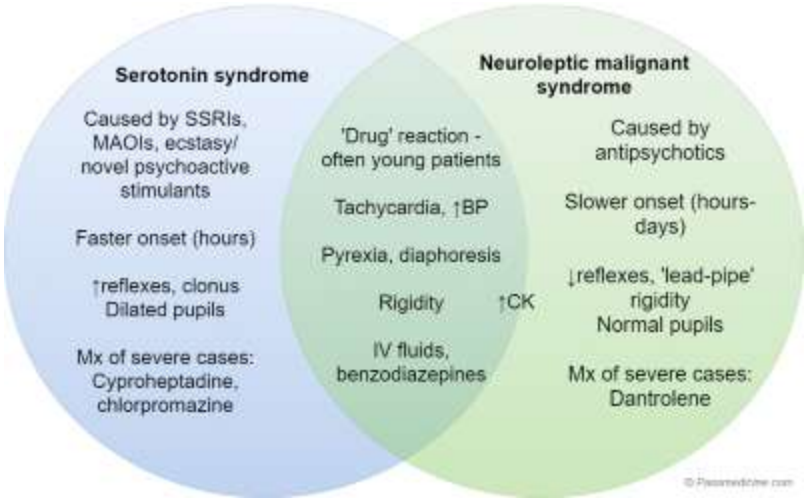
- monoamine oxidase inhibitors
- SSRIs
  - St John's Wort, often taken over the counter for depression, can interact with SSRIs to cause serotonin syndrome
  - tramadol may also interact with SSRIs
- ecstasy
- amphetamines

Features

- neuromuscular excitation
  - hyperreflexia
  - myoclonus
  - rigidity
- autonomic nervous system excitation
  - hyperthermia
  - sweating
- altered mental state
  - confusion

Management

- supportive including IV fluids
- benzodiazepines
- more severe cases are managed using serotonin antagonists such as cyproheptadine and chlorpromazine



Venn diagram showing contrasting serotonin syndrome with neuroleptic malignant syndrome. Note that both conditions can cause a raised creatine kinase (CK) but it tends to be more associated with NMS.

123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medsafe

1 1

Neuroleptice malignant syndrome Vs serotonin syndrome

Suggest link

Report broken link

Media

Serotonin syndrome

Kloss and Bruce - YouTube 7 0

Serotonin Syndrome Explained

Rhesus Medicine - YouTube 3 0

Report broken media

Score: **18.1%**

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Back to top

A 30-year-old man attends the emergency department with chest pain. He describes the pain as a squeezing sensation in the centre of his chest. There is no radiation of his pain but he feels mildly breathless. He works as a city lawyer which he admits can be particularly stressful. He is an occasional smoker and binge-drinks alcohol on the weekends. He has no other past medical history.

On examination, he is tremulous and sweating profusely. He has a temperature of 37.9ºC. His heart rate is 140bpm and regular with a blood pressure of 143/87mmHg. His chest is clear with normal heart sounds. Saturations are 96% on air. He has increased rigidity in his upper and lower limbs. His pupils are dilated and reactive to light.

Investigations:

Hb	151 g/L	Male: (135-180) Female: (115 - 160)
Platelets	288 * 10^9/L	(150 - 400)
WBC	9.1 * 10^9/L	(4.0 - 11.0)
Na^+	149 mmol/L	(135 - 145)
K^+	5.4 mmol/L	(3.5 - 5.0)
Urea	10.3 mmol/L	(2.0 - 7.0)
Creatinine	134 µmol/L	(55 - 120)
D-Dimer	398 ng/L	(<400)
Troponin	67 ng/L	(0-14)

ECG - ST depression in I, aVL, V5 and V6.

He is diagnosed with an NSTEMI and dual antiplatelet therapy, fondaparinux and sublingual glyceryl trinitrate (GTN) is initiated for acute coronary syndrome (ACS). His pain begins to settle after GTN.

What is the most appropriate medication to give in addition to the ACS protocol?

- Atenolol
- Sodium nitroprusside

- Diazepam
- Intravenous fluids
- Glyceryl trinitrate infusion

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

A 30-year-old man attends the emergency department with chest pain. He describes the pain as a squeezing sensation in the centre of his chest. There is no radiation of his pain but he feels mildly breathless. He works as a city lawyer which he admits can be particularly stressful. He is an occasional smoker and binge-drinks alcohol on the weekends. He has no other past medical history.

On examination, he is tremulous and sweating profusely. He has a temperature of 37.9ºC. His heart rate is 140bpm and regular with a blood pressure of 143/87mmHg. His chest is clear with normal heart sounds. Saturations are 96% on air. He has increased rigidity in his upper and lower limbs. His pupils are dilated and reactive to light.

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What is the most appropriate medication to give in addition to the ACS protocol?

Atenolol	15%
Sodium nitroprusside	6%

Diazepam	63%
Intravenous fluids	5%
Glyceryl trinitrate infusion	10%

Cocaine toxicity - avoid beta-blockers

Important for me Less important

This man is likely to have taken a cocaine overdose. Symptoms include an increased sympathetic drive causing hyperthermia, sweating, tachycardia and dilated pupils. Chest pain is secondary to vasospasm of the coronary arteries causing ECG changes and raised cardiac enzymes. Benzodiazepines are indicated in cocaine toxicity and in this scenario, ACS treatment should be initiated with the addition of benzodiazepines such as diazepam.

Sodium nitroprusside is an indicated additive in the instance of cocaine toxicity and hypertension.

Glyceryl trinitrate infusions are indicated in ACS with ongoing chest pain. This patient has shown good response to sublingual GTN, making a GTN infusion less appropriate at this stage.

Intravenous fluids are likely to also be prescribed to treat his co-existing acute kidney injury. However, in the instance of cocaine toxicity and chest pain, diazepam is the more appropriate initial choice.

Atenolol and other beta blockers should be avoided or used with caution in cocaine toxicity due to the risk of unopposed alpha-agonism. Benzodiazepines should be given instead, making diazepam the correct answer.

Discuss (1) Improve

Next question >

Cocaine

Cocaine is an alkaloid derived from the coca plant. It is widely used as a recreational stimulant. The price of cocaine has fallen sharply in the past decade resulting in cocaine toxicity becoming a much more frequent clinical problem. This increase has made cocaine a favourite topic of question writers.

Mechanism of action

- cocaine blocks the uptake of dopamine, noradrenaline and serotonin

Adverse effects

- cardiovascular
  - coronary artery spasm → myocardial ischaemia/infarction
  - both tachycardia and bradycardia may occur
  - hypertension
  - QRS widening and QT prolongation
  - aortic dissection
- neurological
  - seizures
  - mydriasis
  - hypertonia
  - hyperreflexia
- psychiatric effects
  - agitation
  - psychosis
  - hallucinations
- others
  - ischaemic colitis is recognised in patients following cocaine ingestion. This should be considered if patients complain of abdominal pain or rectal bleeding
  - hyperthermia
  - metabolic acidosis
  - rhabdomyolysis

Management of cocaine toxicity

- in general, benzodiazepines are generally first-line for most cocaine-related problems
- chest pain:
  - benzodiazepines + glyceryl trinitrate
  - if myocardial infarction develops then primary percutaneous coronary intervention
- hypertension: benzodiazepines + sodium nitroprusside
- the use of beta-blockers in cocaine-induced cardiovascular problems is a controversial issue
  - the American Heart Association issued a statement in 2008 warning against the use of beta-blockers (due to the risk of unopposed alpha-mediated coronary vasospasm) but many cardiologists since have questioned whether this is valid
  - if a reasonable alternative is given in an exam it is probably wise to choose it

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Cocaine Dependence

Osmosis - YouTube 4 2

Report broken media

Score: **18.1%**

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Back to top

A 35-year-old farmer who is in financial difficulties presents to the Emergency department some 40 minutes after taking an unknown amount of paraquat-based weed killer. On arrival in the Emergency department, he has a Glasgow coma scale score of 15 and tells you he regrets what he has done. He feels sick and says there is a burning sensation in his mouth. On examination his blood pressure is 135/80 mmHg, his pulse is 85 beats per minute and regular. There are no abnormal physical signs. Which of the following is the most important intervention?

- Administration of emetic
- Gastric lavage
- IV normal saline
- IV sodium bicarbonate
- Oral fuller's earth

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

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Administration of emetic	2%
Gastric lavage	42%
IV normal saline	7%
IV sodium bicarbonate	9%
Oral fuller's earth	**40%**

Both activated charcoal and fuller's earth are recognised to adsorb paraquat in vitro. They are thought to be most effective when given in the first two hours after overdose, although benefit may still be present up to 12hrs after ingestion. Neither has significant toxicity issues.

Gastric lavage is not recommended. Administration of emetic risks spreading caustic material further, potentially driving greater absorption of paraquat. IV normal saline and IV sodium bicarbonate do not affect prognosis in paraquat overdose. No anti-inflammatory, immunosuppressive, or anti-oxidant interventions have demonstrated a benefit either.

Discuss (7) | Improve

Next question

Overdose and poisoning: management

The table below outlines the main management for common overdoses:

Toxin	Treatment
Paracetamol	Management • activated charcoal if ingested < 1 hour ago • N-acetylcysteine (NAC) • liver transplantation
Salicylate	Management • urinary alkalinization with IV bicarbonate • haemodialysis
Opioid/opiates	Naloxone
Benzodiazepines	Flumazenil The majority of overdoses are managed with supportive care only due to the risk of seizures with flumazenil. It is generally only used with severe or iatrogenic overdoses.
Tricyclic antidepressants	Management • IV bicarbonate may reduce the risk of seizures and arrhythmias in severe toxicity • arrhythmias: class 1a (e.g. Quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation. Class III drugs such as amiodarone should also be avoided as they prolong the QT interval. Response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in management of tricyclic induced arrhythmias • dialysis is ineffective in removing tricyclics
Lithium	Management • mild-moderate toxicity may respond to volume resuscitation with normal saline • haemodialysis may be needed in severe toxicity • sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion
Warfarin	Vitamin K, prothrombin complex
Heparin	Protamine sulphate
Beta-blockers	Management • if bradycardic then atropine

Toxin	Treatment
	• in resistant cases glucagon may be used
Ethylene glycol	Management has changed in recent times • ethanol has been used for many years • works by competing with ethylene glycol for the enzyme alcohol dehydrogenase • this limits the formation of toxic metabolites (e.g. Glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning • **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol • haemodialysis also has a role in refractory cases
Methanol poisoning	Management • fomepizole or ethanol • haemodialysis
Organophosphate insecticides	Management • atropine • the role of pralidoxime is still unclear - meta-analyses to date have failed to show any clear benefit
Digoxin	Digoxin-specific antibody fragments
Iron	Desferrioxamine, a chelating agent
Lead	Dimercaprol, calcium edetate
Carbon monoxide	Management • 100% oxygen • hyperbaric oxygen
Cyanide	Hydroxocobalamin; also combination of amyl nitrite, sodium nitrite, and sodium thiosulfate

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

BNF

Emergency treatment of poisoning

2 1

Royal College of Physicians

2012 Poisoning and self-harm update

1 1

Royal College of Physicians

2007 Acute poisoning on the medical admissions unit

1 2

Suggest link

Report broken link

Score: **18.1%**

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A 63-year-old woman is brought in by ambulance to the emergency department, having been found at home by her neighbour with a decreased level of consciousness. She has a past medical history of osteoarthritis and hypertension. She takes ramipril and uses ibuprofen regularly for pain relief.

Her observations are heart rate of 118 beats per minute, blood pressure 95/66mmHg, respiratory rate of 20/min, temperature 36.7ºC and oxygen saturations of 94% on room air.

Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable. Her Glasgow coma scale is 11/15 (E3, V3, M5).

IV fluids are administered and an arterial blood gas and blood tests are obtained.

Arterial blood gas results:

pH	7.24	(7.35-7.45)
paO2	10.2 kPa	(10-14)
paCO2	5.7 kPa	(4.5 - 6.0
Base excess	-4.2 mmol/L	(-2 - +2)
Chloride	95 mmol/L	(95-105)
Bicarbonate	14 mmol/L	(22-26)
Glucose	5.0 mmol/L	(4-11)
Lactate	2.5 mmol/L	(0.5-2.0)
SaO2	94 %	(94-98)

Blood test results:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.4 * 10^9/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	5.0 mmol/L	(3.5 - 5.0)

Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	324 mOsm/Kg	(280-290)

What is the most likely diagnosis?

- ○ Aspirin overdose ×
- ○ Ethylene glycol administration ×
- ○ IV sodium chloride 0.9% administration ×
- ○ NSAID treatment ×
- ○ Ramipril overdose ×

Submit answer

Reference ranges

Score: **15.1%**

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A 63-year-old woman is brought in by ambulance to the emergency department, having been found at home by her neighbour with a decreased level of consciousness. She has a past medical history of osteoarthritis and hypertension. She takes ramipril and uses ibuprofen regularly for pain relief.

Her observations are heart rate of 118 beats per minute, blood pressure 95/66mmHg, respiratory rate of 20/min, temperature 36.7ºC and oxygen saturations of 94% on room air.

Cardiovascular, respiratory and abdominal examinations are otherwise unremarkable. Her Glasgow coma scale is 11/15 (E3, V3, M5).

IV fluids are administered and an arterial blood gas and blood tests are obtained.

Arterial blood gas results:

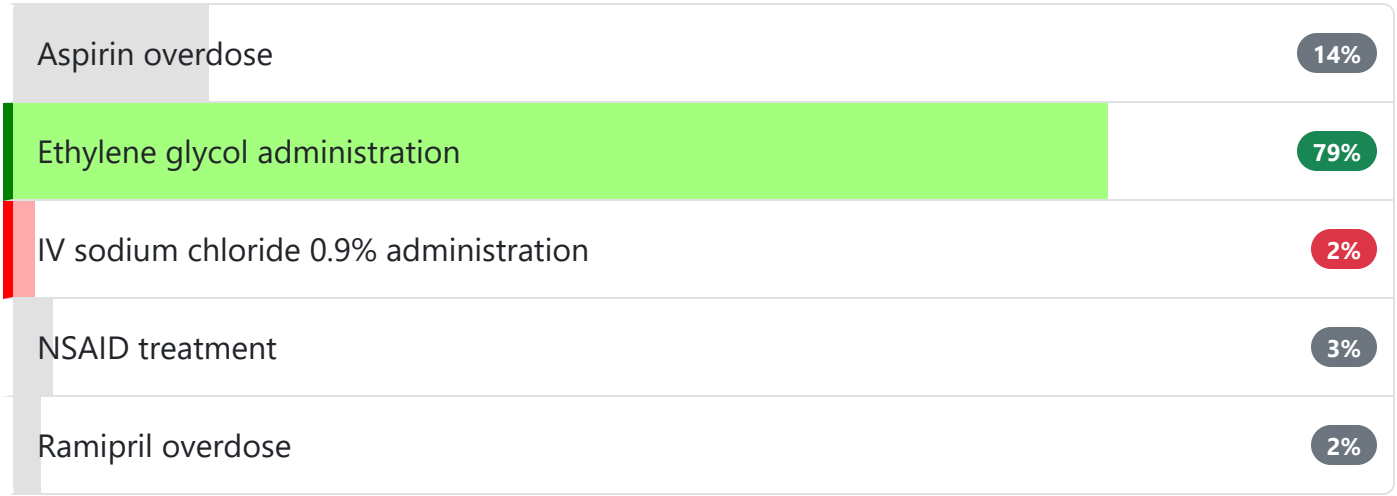
pH	7.24	(7.35-7.45)
paO2	10.2 kPa	(10-14)
paCO2	5.7 kPa	(4.5 - 6.0
Base excess	-4.2 mmol/L	(-2 - +2)
Chloride	95 mmol/L	(95-105)
Bicarbonate	14 mmol/L	(22-26)
Glucose	5.0 mmol/L	(4-11)
Lactate	2.5 mmol/L	(0.5-2.0)
SaO2	94 %	(94-98)

Blood test results:

Hb	137 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10^9/L	(150 - 400)
WBC	4.4 * 10^9/L	(4.0 - 11.0)
Na^+	141 mmol/L	(135 - 145)
K^+	5.0 mmol/L	(3.5 - 5.0)

Urea	8.0 mmol/L	(2.0 - 7.0)
Creatinine	111 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Measured osmolality	324 mOsm/Kg	(280-290)

What is the most likely diagnosis?



Metabolic acidosis with a high anion gap and high osmolar gap is seen in ethylene glycol poisoning

Important for me Less important

Ethylene glycol administration is the correct answer. This is a case of a raised anion gap metabolic acidosis with a raised osmolar gap. The anion gap is calculated as (sodium + potassium) - (chloride + bicarbonate). In this case, it is 37 mmol/L, which is raised (normal range 8-16 mmol/L). The osmolar gap is calculated as osmolality (measured) - osmolarity (calculated). Osmolarity (calculated) is [2*sodium + glucose + urea]. In this case, it is (324) - (295) = 29mOsm (normal range < 10), indicating a raised osmolar gap. A high osmolar gap means the presence of an unmeasured solute in high amounts e.g. ethylene glycol in this case.

Aspirin overdose is incorrect. This can cause a high anion gap metabolic acidosis with a high osmolar gap. However, If the aspirin overdose was sufficiently large to cause the patient to be obtunded with a low GCS, we would expect a respiratory alkalosis and possibly convulsions. Aspirin has direct effects on the respiratory centre and causes tachypnoea and respiratory alkalosis. In this case, the oxygen saturations are 94%, the pO2 is low normal, the pCO2 is on the upper end of normal and the respiratory rate is merely 20, making this a less likely cause clinically than ethylene glycol.

IV sodium chloride 0.9% administration is incorrect. This causes a normal anion gap metabolic acidosis with high chloride. Additionally, the fluids were administered after she had become unwell, excluding this as a cause of her presentation.

NSAID treatment is incorrect. This can cause a type 1 renal tubular acidosis, which will have a normal anion gap.

Ramipril overdose is incorrect. This is associated with a type 4 renal tubular acidosis, which will have a normal anion gap. Furthermore, if the patient took a significant ramipril overdose, we might expect the blood pressure to be lower.

Discuss (3) | Improve

Next question >

Ethylene glycol toxicity

Ethylene glycol is a type of alcohol used as a coolant or antifreeze

Features of toxicity are divided into 3 stages:

- Stage 1: symptoms similar to alcohol intoxication: confusion, slurred speech, dizziness
- Stage 2: metabolic acidosis with high anion gap and high osmolar gap. Also tachycardia, hypertension
- Stage 3: acute kidney injury

Management has changed in recent times

- ethanol has been used for many years
- works by competing with ethylene glycol for the enzyme alcohol dehydrogenase
- this limits the formation of toxic metabolites (e.g. glycoaldehyde and glycolic acid) which are responsible for the haemodynamic/metabolic features of poisoning
- **fomepizole**, an inhibitor of alcohol dehydrogenase, is now used first-line in preference to ethanol
- haemodialysis also has a role in refractory cases

123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Life in the Fast Lane

Toxic Alcohol Ingestion

4 1

Suggest link

Report broken link

Score: **18.1%**

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His past medical history includes hypertension, mild chronic kidney disease and atrial fibrillation.

He is currently on Ramipril 2.5mg once daily, Bisoprolol 5mg once daily, and treatment dose Clexane (patient was on Warfarin pre-operatively).

Pre-admission operative bloods:

Hb	142 g/l
Platelets	212 * 10^9/l
WBC	14.1 * 10^9/l

Na^+	138 mmol/l
K^+	4.6 mmol/l
Urea	8.2 mmol/l
Creatinine	167 µmol/l

Bloods yesterday:

Hb	112 g/l
Platelets	27 * 10^9/l
WBC	10.1 * 10^9/l

Na^+	136 mmol/l
K^+	4.0 mmol/l
Urea	9.1 mmol/l
Creatinine	182 µmol/l

What should you do with regards to his Clexane?

- Stop Clexane and give Warfarin (target INR 2-3)
- Stop Clexane and give treatment dose Argatroban
- Switch treatment dose Clexane to Unfractionated Heparin infusion
- Give 1 pool of platelets
- Switch treatment dose Clexane to prophylactic Clexane

Submit answer

Reference ranges

Score: **15.1%**

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Urea	9.1 mmol/l
Creatinine	182 µmol/l

What should you do with regards to his Clexane?

Stop Clexane and give Warfarin (target INR 2-3)	5%
Stop Clexane and give treatment dose Argatroban	78%
Switch treatment dose Clexane to Unfractionated Heparin infusion	12%
Give 1 pool of platelets	3%
Switch treatment dose Clexane to prophylactic Clexane	2%

This is a case of heparin induced thrombocytopaenia

Criteria	**2**	**1**	**0**
Thrombocytopaenia	>50% fall, and nadir >$20*10^9$/l	30-50% fall, or nadir 10-$19*10^9$/l	fall < 30%, or nadir < $10*10^9$/l
Timing of platelet fall	Between 5-10 days, or <1 day if exposure to heparin within last 20 days	consistent with immunisation, unclear due to missing samples	fall <4 days after exposure (with no recent exposure history
Sequelae	Thrombosis, skin necrosis or systemic reaction post-bolus	Progressive or recurrent thrombosis, suspected but unproven thrombosis, erythematous skin reaction	
Alternative cause	None	Possible	Definite

0-3 - Low
4-5 - Intermediate
6-8 - High

This patient fits all the criteria for heparin induced thrombocytopenia

The recommendations for treatment are as follows:

- Therapeutic danaparoid should be used
- The anticoagulant effect of danaparoids can be measured with anti-Xa assay
- Therapeutic dose fondaparinux is an acceptable alternative anticoagulant (although it is unlicensed)
- Therapeutic anticoagulation should be continued for 3 months (in those with thrombosis) and 4 weeks (in those without thrombosis)

- When transitioning from argatroban to warfarin, the INR should be >4 for 2 days prior to discontinuing argatroban
- Warfarin should not be used till platelet count is back in normal range

British Society of Haematology:
http://onlinelibrary.wiley.com/doi/10.1111/bjh.12059/epdf

Discuss (9) Improve

Next question >

Heparin ★

There are two main types of heparin - unfractionated, 'standard' heparin or low molecular weight heparin (LMWH). Heparins generally act by activating antithrombin III. Unfractionated heparin forms a complex which inhibits thrombin, factors Xa, IXa, XIa and XIIa. LMWH however only increases the action of antithrombin III on factor Xa

Adverse effects of heparins include:

- bleeding
- thrombocytopenia - see below
- osteoporosis and an increased risk of fractures
- hyperkalaemia - this is thought to be caused by inhibition of aldosterone secretion

The table below shows the differences between standard heparin and LMWH:

	Standard heparin	**Low molecular weight heparin (LMWH)**
Administration	Intravenous	Subcutaneous
Duration of action	Short	Long
Mechanism of action	Activates antithrombin III. Forms a complex that inhibits thrombin, factors Xa, IXa, Xia and XIIa	Activates antithrombin III. Forms a complex that inhibits factor Xa
Side-effects	Bleeding Heparin-induced thrombocytopaenia	Bleeding

	Standard heparin	Low molecular weight heparin (LMWH)
	(HIT) Osteoporosis	Lower risk of HIT and osteoporosis with LMWH
Monitoring	Activated partial thromboplastin time (APTT)	Anti-Factor Xa (although routine monitoring is not required)
Notes	Useful in situations where there is a high risk of bleeding as anticoagulation can be terminated rapidly. Also useful in renal failure	Now standard in the management of venous thromboembolism treatment and prophylaxis and acute coronary syndromes

Heparin-induced thrombocytopaenia (HIT)

- immune mediated - antibodies form against complexes of platelet factor 4 (PF4) and heparin
- these antibodies bind to the PF4-heparin complexes on the platelet surface and induce platelet activation by cross-linking FcγIIA receptors
- usually does not develop until after 5-10 days of treatment
- despite being associated with low platelets HIT is actually a prothrombotic condition
- features include a greater than 50% reduction in platelets, thrombosis and skin allergy
- address need for ongoing anticoagulation:
  - direct thrombin inhibitor e.g. argatroban
  - danaparoid

Heparin overdose may be reversed by protamine sulphate, although this only partially reverses the effect of LMWH.

AI + Q 123

Next question >

B I A T↕

Textbooks

High-yield textbook

Extended textbook

Links

BNF 3 2

Heparin

British Society for Haematology 2 2

2012 HIT guidelines

Suggest link Report broken link

Media

Heparin-induced thrombocytopenia

Osmosis - YouTube 7 0

Report broken media

Score: **18.1%**

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- Recent addition of carbamazepine to augment lithium
- Recent prescription of naproxen for knee pain
- Recent increase in caffeine intake
- Recent addition of amiodarone for atrial fibrillation
- Recent use of sodium bicarbonate tablets for symptoms of dyspepsia

Submit answer

Reference ranges

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Recent addition of carbamazepine to augment lithium	17%
Recent prescription of naproxen for knee pain	57%
Recent increase in caffeine intake	3%
Recent addition of amiodarone for atrial fibrillation	19%
Recent use of sodium bicarbonate tablets for symptoms of dyspepsia	4%

The answer is B. NSAIDs reduce excretion of lithium in the kidneys and can, therefore, increase serum lithium levels and give rise to lithium toxicity as seen above. ACE inhibitors, ARBs and thiazide diuretics also reduce the kidney's ability to excrete lithium.

Caffeine and sodium bicarbonate both increase the excretion of lithium from the body and therefore will reduce serum lithium levels

When carbamazepine is added to lithium therapy, it is known to increase the risk of neurotoxicity, but it does this without increasing serum lithium levels. Similarly, use of amiodarone with lithium is known to increase the risk of VT, but it does this without increasing lithium levels

Discuss (3) | Improve

Next question

Lithium toxicity

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure
- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- hyperreflexia
- acute confusion
- polyuria
- seizure
- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this
  - by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 25-year-old female was admitted to the emergency department following an overdose of unknown tablets 4 hours ago. Her past medical history includes depression, previous overdoses and gastric ulcer disease. On examination, she was drowsy but easily rousable with a Glasgow Coma Score of 14. She has a heart rate of 112 beats per minute, blood pressure of 106/60mmHg, respiratory rate of 22 and saturations of 96% on air.

Electrocardiogram shows a sinus tachycardia with QRS complexes measuring 160 ms.

An arterial blood gas on air revealed the following results:

pH	7.29
pCO2	6.2 kPa
pO2	10.5 kPa
HCO3-	18 mmol/l
BE	-6.6 mmol/l

Which of the following management is the most appropriate?

- Intravenous magnesium
- Activated charcoal
- Gastric lavage
- Sodium bicarbonate
- 5% dextrose infusion

Submit answer

Reference ranges

Score: **15.1%**

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pH	7.29
pCO2	6.2 kPa
pO2	10.5 kPa
HCO3-	18 mmol/l
BE	-6.6 mmol/l

Which of the following management is the most appropriate?

Intravenous magnesium	5%
Activated charcoal	3%
Gastric lavage	1%
Sodium bicarbonate	89%
5% dextrose infusion	0%

Tricyclic Antidepressant (TCA) overdose is most likely in this case, based on the presentation, mixed acidosis seen on ABG and ECG changes. TCA overdose can present in various ways. Cardiovascular effects include tachycardia, prolong QRS complexes and induce cardiac arrhythmias. Central nervous system effects include altered mental status, coma and seizures.

- Intravenous magnesium: no indications in this case of TCA overdose
- Activated charcoal: only appropriate if GCS is not reduced and if patients present within 2 hours of ingestion

- Gastric lavage: may be considered for potentially life-threatening TCA overdoses only when it can be delivered within 1h of ingestion and the airway is protected
- Sodium bicarbonate: is the main treatment for dysrhythmias or hypotension associated with TCA overdoses
- 5% dextrose infusion: has no role in treatment of TCA poisoning or fluid resuscitation

Source: Guidelines in Emergency Medicine Network (GEMNet): guideline for the management of tricyclic antidepressant overdose

Discuss (3) | Improve

Next question >

Tricyclic overdose ★

Overdose of tricyclic antidepressants is a common presentation to emergency departments. Amitriptyline and dosulepin (dothiepin) are particularly dangerous in overdose.

Early features relate to anticholinergic properties: dry mouth, dilated pupils, agitation, sinus tachycardia, blurred vision.

Features of severe poisoning include:
- arrhythmias
- seizures
- metabolic acidosis
- coma

ECG changes include:
- sinus tachycardia
- widening of QRS
- prolongation of QT interval

Widening of QRS > 100ms is associated with an increased risk of seizures whilst QRS > 160ms is associated with ventricular arrhythmias

Management

- IV bicarbonate
  - first-line therapy for hypotension or arrhythmias
  - indications include widening of the QRS interval >100 msec or a ventricular arrhythmia
- other drugs for arrhythmias

+ class 1a (e.g. quinidine) and class Ic antiarrhythmics (e.g. Flecainide) are contraindicated as they prolong depolarisation

- class III drugs such as amiodarone should also be avoided as they prolong the QT interval
- response to lignocaine is variable and it should be emphasized that correction of acidosis is the first line in the management of tricyclic-induced arrhythmias

- intravenous lipid emulsion is increasingly used to bind free drug and reduce toxicity
- dialysis is ineffective in removing tricyclics

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 40-year-old woman is brought into the emergency department after being found unresponsive by her partner. Her partner reports that she has been well up until today. She has a past medical history of fibromyalgia, migraines and depression for which she has tried several pharmacological agents.

On examination, she has a heart rate of 120 beats/min with a blood pressure of 131/76mmHg. Her chest sounds are clear with a respiratory rate of 28 breaths/min and oxygen saturations of 94% on air. She is drowsy but responsive to voice and her pupils are dilated. She smells strongly of alcohol.

An ECG is taken:

© Image used on license from Dr Smith, University of Minnesota

Given the likely diagnosis, what is the recommended management for this patient?

- DC cardioversion
- Fomepizole
- Haemofiltration
- Magnesium sulphate
- Sodium bicarbonate

Submit answer

Reference ranges

Score: **15.1%**

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An ECG is taken:

© Image used on license from Dr Smith, University of Minnesota

Given the likely diagnosis, what is the recommended management for this patient?

DC cardioversion	3%
Fomepizole	28%
Haemofiltration	4%
Magnesium sulphate	10%
Sodium bicarbonate	55%

Sodium bicarbonate is correct. This patient has a likely diagnosis of a tricyclic antidepressant (TCA) overdose given the tachycardia and dilated pupils in combination with this patient's ECG. This patient's ECG shows tachycardia with widened QRS complexes which is a characteristic feature

of a TCA overdose. The past medical history of depression is another suggestion that this patient may be prescribed tricyclic antidepressants. The medical management includes intravenous sodium bicarbonate, particularly if there are any arrhythmias or QRS widening on the ECG. Of note, a QRS interval > 160ms is associated with an increased risk of arrhythmias (e.g. ventricular fibrillation).

DC cardioversion is incorrect. This is used in the cardioversion of tachyarrhythmias with adverse features (e.g. ischaemia, shock, syncope and heart failure). In patients with a TCA overdose, DC cardioversion is unlikely to be successful even in the presence of a ventricular arrhythmia. In this circumstance, intravenous sodium bicarbonate is still recommended along with supporting any additional features (e.g. intubation for low GCS or IVF/phenylephedrine for hypotension).

Fomepizole is incorrect. This is used in the management of ethylene glycol and methanol poisoning. Although this patient smells strongly of alcohol which is suggestive of ethylene glycol or methanol use, the past medical history of depression and the patient's ECG findings are more likely to be secondary to a TCA overdose.

Haemofiltration is incorrect. Although haemofiltration can remove some substances from the circulation (e.g. barbituates, lithium, alcohol, salicylates and theophylline), it has no role in the management of a TCA overdose.

Magnesium sulphate is incorrect. This is used in the management of torsades de pointes, a polymorphic ventricular tachycardia associated with a widened QTc interval. This arrhythmia is more likely to be present if the patient had taken an overdose of a serotonin-selective reuptake inhibitor (SSRI) which is also used to treat depression. However, this arrhythmia is not what is seen here making magnesium sulphate incorrect.

Discuss (1) | Improve

Next question >

Tricyclic overdose

Overdose of tricyclic antidepressants is a common presentation to emergency departments. Amitriptyline and dosulepin (dothiepin) are particularly dangerous in overdose.

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AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 58-year-old gentleman is admitted to the acute medical unit with abnormal movements and confusion. He has a past medical history of bipolar disorder and epilepsy. His drug history includes lithium and sodium valproate. On examination you note choreathetoid movements of all four limbs. His Glasgow coma score is 11 (M5, E3, V3).

Blood results are as follows:

Hb	145 g/l	Na^+	138 mmol/l
Platelets	524 * 10^9/l	K^+	3.9 mmol/l
WBC	11.6 * 10^9/l	Urea	10.5 mmol/l
Neuts	9.8 * 10^9/l	Creatinine	125 µmol/l
Lithium level	2.6 mmol/L (therapeutic range 0.4 to 1.0 mmol/L)		

What definitive treatment does this patient require?

- Sodium bicarbonate
- Gastric lavage
- Haemodialysis
- Whole bowel irrigation
- Reassurance

Submit answer

Reference ranges

Score: **15.1%**

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Lithium level	2.6 mmol/L (therapeutic range 0.4 to 1.0 mmol/L)		

What definitive treatment does this patient require?

Acute on chronic overdose of lithium can lead to serious toxicity even after a modest overdose, as the the extracellular tissues are already saturated with lithium

Important for me Less important

Acute on chronic overdose of lithium can lead to serious toxicity even after a modest overdose, as the extracellular tissues are already saturated with lithium.

Dialysis should be considered in symptomatic lithium toxicity and:

- A lithium concentration greater than 5.0 mmol/L in patients with acute lithium overdose and who are not prescribed lithium

- A lithium concentration greater than 2.5 mmol/L in patients with chronic poisoning

Haemodialysis should be continued until:

- All new neurological features have resolved, and
- Lithium concentration remains stable at less than 1mmol/L

Gastric lavage, even if possible within one hour of overdose, is likely to be of limited value as slow-release tablets do not disintegrate in the stomach and most are too large to pass up to a lavage tube.

Whole bowel irrigation can be used, however, there are no clinical trials to confirm its efficacy.

Some studies have shown that urinary alkalisation with sodium bicarbonate may be of use, however the evidence is weak.

Ultimately, this patient has severe neurological features, and a lithium level > 2.5 mmol/L in the context of chronic poisoning, and therefore the definitive management required is haemodialysis. The patient should be fluid resuscitated with crystalloid while discussing the case with a renal physician to arrange dialysis.

Discuss (4) | Improve

Next question >

Lithium toxicity ★

Lithium is a mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.4-1.0 mmol/L) and a long plasma half-life being excreted primarily by the kidneys. Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by:

- dehydration
- renal failure
- drugs: diuretics (especially thiazides), ACE inhibitors/angiotensin II receptor blockers, NSAIDs and metronidazole.

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)

- hyperreflexia
- acute confusion
- polyuria
- seizure
- coma

Management

- mild-moderate toxicity may respond to volume resuscitation with normal saline
  - IV fluids with isotonic saline, until euvolemic, then typically twice maintenance rate
  - monitor serum sodium closely (every 4 hours with serial lithium concentrations) if there is a concern about lithium-induced nephrogenic diabetes insipidus
- haemodialysis may be needed in severe toxicity
- sodium bicarbonate is sometimes used but there is limited evidence to support this
  - by increasing the alkalinity of the urine it promotes lithium excretion

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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Back to top

A 42-year-old man presents to his GP, wanting to discuss his alcohol intake. For several years, he has been drinking 15-20 units per day. He is now keen to cut down to recommended limits but is enquiring about potential pharmacological options that can be used to aid him.

What can be used to promote abstinence by causing unpleasant symptoms?

- Acamprosate
- Diazepam
- Disulfiram
- Naltrexone
- Thiamine

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

A 42-year-old man presents to his GP, wanting to discuss his alcohol intake. For several years, he has been drinking 15-20 units per day. He is now keen to cut down to recommended limits but is enquiring about potential pharmacological options that can be used to aid him.

What can be used to promote abstinence by causing unpleasant symptoms?

Acamprosate	23%
Diazepam	3%
Disulfiram	70%
Naltrexone	3%
Thiamine	2%

Disulfram is an acetaldehyde dehydrogenase inhibitor. When combined with alcohol, it results in high levels of acetaldehyde which can cause flushing of the skin, nausea, vomiting, arrhythmias, and even death

Important for me Less important

The correct answer is **disulfiram**. This is an acetaldehyde dehydrogenase inhibitor. By inhibiting this enzyme, consumption of alcohol leads to a build-up of acetaldehyde, which can cause unpleasant symptoms such as flushing of the skin, nausea, vomiting, and arrhythmias. However, a problem with the use of disulfiram is poor compliance, as it does not reduce cravings for alcohol.

Acamprosate is another drug used to promote abstinence. Rather than causing unpleasant symptoms, it reduces the craving for alcohol. It is a weak antagonist of NMDA receptors.

Diazepam is incorrect. Whilst this benzodiazepine is often used in the management of acute withdrawal, it does not cause symptoms like disulfiram.

Naltrexone is incorrect. This is used to treat both opioid and alcohol dependence. Rather than causing unpleasant symptoms, however, it instead reduces cravings and feelings of euphoria associated with substance use disorder.

Thiamine is also incorrect. This is recommended to be given orally to alcoholic patients if their diet may be deficient. It does not cause unpleasant symptoms.

Discuss (3) | Improve

Next question

Alcohol - problem drinking: management

Nutritional support

- SIGN recommends alcoholic patients should receive oral thiamine if their 'diet may be deficient'

Drugs used

- benzodiazepines for acute withdrawal
- disulfram: promotes abstinence - alcohol intake causes severe reaction due to inhibition of acetaldehyde dehydrogenase. Patients should be aware that even small amounts of alcohol (e.g. In perfumes, foods, mouthwashes) can produce severe symptoms. Contraindications include ischaemic heart disease and psychosis
- acamprosate: reduces craving, known to be a weak antagonist of NMDA receptors, improves abstinence in placebo controlled trials

AI + Q 123

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Clinical Knowledge Summaries

1 1

Alcohol - problem drinking

NICE

3 3

2011 Alcohol-use disorders

Suggest link Report broken link

Media

Alcoholism

Osmosis - YouTube 1 0

Report broken media

Score: **18.1%**

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Back to top

A 27-year-old lady presents to her GP with symptoms of polyuria and dysuria. She is currently 7 weeks into her second pregnancy. Her urine dipstick shows 2+ protein, 2+ blood, 3+ leucocytes and is positive for nitrates. Her records indicate a past reaction to penicillin. Which of the following would be the most appropriate in her treatment?

- Nitrofurantoin
- Trimethoprim
- Co-amoxiclav
- Cefalexin
- Ciclosporin

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

A 27-year-old lady presents to her GP with symptoms of polyuria and dysuria. She is currently 7 weeks into her second pregnancy. Her urine dipstick shows 2+ protein, 2+ blood, 3+ leucocytes and is positive for nitrates. Her records indicate a past reaction to penicillin. Which of the following would be the most appropriate in her treatment?

Nitrofurantoin	67%
Trimethoprim	11%
Co-amoxiclav	4%
Cefalexin	17%
Ciclosporin	1%

Trimethoprim should be avoided in the first 3 months of pregnancy

Important for me Less important

Nitrofurantoin has been used extensively in the treatment of UTIs in pregnant ladies in first trimester since the 1950s and has a good safety profile. The anti-folate action of trimethoprim could yield trimethoprim as a second-line agent provided that enough folate cover is offered. NICE guidelines state that 5 mg of folate must be co-administered in case that trimethoprim is prescribed. The antibiotic regime unlike simple UTI lasts for 7 days.

Discuss (4) Improve

Next question

Prescribing in pregnant patients

Very few drugs are known to be completely safe in pregnancy. The list below largely comprises of those known to be harmful. Some countries have developed a grading system - see the link.

Antibiotics

- tetracyclines
- aminoglycosides
- sulphonamides and trimethoprim
- quinolones: the BNF advises to avoid due to arthropathy in some animal studies

Other drugs

- ACE inhibitors, angiotensin II receptor antagonists
- statins
- warfarin
- sulfonylureas
- retinoids (including topical)
- cytotoxic agents

The majority of antiepileptics including valproate, carbamazepine and phenytoin are known to be potentially harmful. The decision to stop such treatments however is difficult as uncontrolled epilepsy is also a risk

AI + Q 123

Next question >

Textbooks

High-yield textbook

Extended textbook

Links

Australian goverment

1 0

Prescribing in pregnancy

GP notebook 1 0

Prescribing in pregnancy

UKTIS 1 0

Co-amoxiclav in pregnancy

Suggest link Report broken link

Score: **18.1%**

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Back to top

The patient below has noticed a gradual change to his facial skin since starting a new medication a few months ago:

© Image used on license from DermNet NZ

Which drug has he been taking?

- Amiodarone
- Phenytoin
- Ciclosporin
- Digoxin
- Sildenafil

Submit answer

Reference ranges

Score: **15.1%**

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Back to top

The patient below has noticed a gradual change to his facial skin since starting a new medication a few months ago:

© Image used on license from DermNet NZ

Which drug has he been taking?

Amiodarone	65%
Phenytoin	17%
Ciclosporin	8%
Digoxin	3%
Sildenafil	7%

The correct answer is **Amiodarone**. This drug, which is used to manage arrhythmias, can cause a bluish-grey discolouration of the skin due to the deposition of lipofuscin pigments. This effect is particularly noticeable on sun-exposed areas such as the face and hands. The condition is usually reversible upon discontinuation of the drug, although it may take several months for the skin colour to return to normal.

The other options are incorrect for the following reasons:

Phenytoin can cause a variety of skin changes including hirsutism, acneiform eruptions, and gingival hyperplasia but it does not typically cause skin discolouration.

Ciclosporin, an immunosuppressant agent used in transplant patients and certain autoimmune conditions, can cause hypertrichosis (excessive hair growth) and gingival hyperplasia. It may also increase susceptibility to infections and malignancies due to its immunosuppressive effects. However, it does not lead to skin discolouration.

Digoxin, a cardiac glycoside used in heart failure and atrial fibrillation, has many potential side effects but they do not include skin discolouration. Instead, digoxin toxicity can manifest with visual disturbances (xanthopsia or yellow vision), gastrointestinal symptoms (nausea, vomiting), neurological symptoms (confusion, delirium), and cardiac arrhythmias.

Finally, **Sildenafil**, commonly known as Viagra and used for erectile dysfunction and pulmonary arterial hypertension, does not typically cause any significant changes in skin colour either. Its most common side effects include headache, flushing, dyspepsia, visual disturbances (blurred vision or cyanopsia - seeing blue-tinted vision), nasal congestion or rhinitis.

Discuss (6) Improve

Next question >

Amiodarone: adverse effects

Adverse effects of amiodarone use

- thyroid dysfunction: both hypothyroidism and hyper-thyroidism
- corneal deposits
- pulmonary fibrosis/pneumonitis
- liver fibrosis/hepatitis
- peripheral neuropathy, myopathy
- photosensitivity
- 'slate-grey' appearance
- thrombophlebitis and injection site reactions
- bradycardia
- lengths QT interval

Important drug interactions of amiodarone include:

- decreased metabolism of warfarin, therefore increased INR
- increased digoxin levels

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18.1%**

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A 47-year-old man is admitted to the Medical Assessment Unit with a pneumonia. He has had a cough for the last week which has not settled with amoxicillin from his GP.

His past medical history includes alcoholic liver disease with a hepatic transplant 6 months previously, hypertension and diet-controlled type II diabetes. He has remained abstinent from alcohol for the last 18 months and is a lifelong non-smoker. His medications are ramipril, simvastatin and tacrolimus.

On admission he has a temperature of 38.1 ºC and oxygen saturations of 94% on air. His heart rate is 110 beats per minute and blood pressure is 105/65 mmHg. On auscultation he has left basal crepitations and chest x-ray confirms a left lower lobe pneumonia.

He is started on intravenous fluids, 2 litres oxygen via nasal cannulae, co-amoxiclav and clarithromycin. His statin is withheld.

2 days later, the ward team are asked to review the patient as he has developed limb twitching. During the assessment he has a generalised tonic-clonic seizure which requires lorazepam to terminate it.

Which investigation is most likely to reveal the cause for his seizures?

- Blood cultures
- CT head
- Electrolytes
- Lumbar puncture
- Tacrolimus level

Submit answer

Reference ranges

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A 47-year-old man is admitted to the Medical Assessment Unit with a pneumonia. He has had a cough for the last week which has not settled with amoxicillin from his GP.

His past medical history includes alcoholic liver disease with a hepatic transplant 6 months previously, hypertension and diet-controlled type II diabetes. He has remained abstinent from alcohol for the last 18 months and is a lifelong non-smoker. His medications are ramipril, simvastatin and tacrolimus.

On admission he has a temperature of 38.1 ºC and oxygen saturations of 94% on air. His heart rate is 110 beats per minute and blood pressure is 105/65 mmHg. On auscultation he has left basal crepitations and chest x-ray confirms a left lower lobe pneumonia.

He is started on intravenous fluids, 2 litres oxygen via nasal cannulae, co-amoxiclav and clarithromycin. His statin is withheld.

2 days later, the ward team are asked to review the patient as he has developed limb twitching. During the assessment he has a generalised tonic-clonic seizure which requires lorazepam to terminate it.

Which investigation is most likely to reveal the cause for his seizures?

Blood cultures	0%
CT head	2%
Electrolytes	13%
Lumbar puncture	3%
Tacrolimus level	82%

The first issue to consider in this question is whether this gentleman's seizure is most likely to be related to infection or metabolic disturbance.

Given that it is two days following his admission with infection, it is unlikely that he has an undetected meningitis, as he would likely have exhibited other symptoms and become much more unwell in this time.

A CT head would be done prior to a lumbar puncture but would not confirm a diagnosis of meningitis, and this gentleman is unlikely to have a new intracranial pathology given the history. Although febrile seizures are a possibility, they are unusual in adults, and again would be uncommon after 2 days of appropriate antibiotics.

Although blood cultures would of course have been taken, they would not prove that a seizure was the result of infection.

This leaves metabolic causes, including hypoglycaemia, hypomagnesaemia, hyper- and hyponatraemia and an altered tacrolimus level.

Tacrolimus is metabolised by the cytochrome P450 system of enzymes. This gentleman has been started on clarithromycin as an antibiotic, which inhibits this system and is therefore likely to cause an increased blood concentration of tacrolimus. Side effects of high tacrolimus level include hyperkalaemia, hypomagnesaemia, hyperglycaemia, hypertension, twitching and seizures. Although checking electrolytes may give clues as to the cause of this gentleman's seizure, and should definitely be done, a tacrolimus level is necessary to prove the diagnosis.

It is also worth keeping in mind that in this type of patient, the suspicion of withdrawal seizures may be high. However, one would expect to see other signs and symptoms earlier on in the admission.

Reference: British National Formulary

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Tacrolimus ★

Tacrolimus is a calcineurin inhibitor used as an immunosuppressant to prevent transplant rejection. It has a very similar action to ciclosporin.

Mechanism of action

- decreases clonal proliferation of T cells by reducing IL-2 release
- binds to FKBP forming a complex which inhibits calcineurin, a phosphotase that activates various transcription factors in T cells
    - this contrasts with ciclosporin, which binds to cyclophilin rather than FKBP

Tacrolimus is more potent than ciclosporin and hence the incidence of organ rejection is less. However, nephrotoxicity and impaired glucose tolerance is more common

Textbooks

High-yield textbook

Extended textbook

Media

Calcineurin Inhibitors (Tacrolimus and Cyclosporine) IL2 - Mechanism of action, adverse effects

Armando Hasudungan - YouTube

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Report broken media

Score: **18.1%**

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